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CONTRACT NUMBER DAMD17-91-C-1120

TITLE: Histological Evaluation of Inner Ears

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CONTRACTING ORGANIZATION: The Research Foundation of the  
State University of New York  
Albany, New York 12201-0009

REPORT DATE: July 1996

TYPE OF REPORT: Final

PREPARED FOR: U.S. Army Medical Research and Materiel Command  
Fort Detrick, Maryland 21702-5012

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Form Approved  
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1. AGENCY USE ONLY (Leave blank)		2. REPORT DATE July 1996	3. REPORT TYPE AND DATES COVERED Final (1 Jul 91 - 30 Jun 96)	
4. TITLE AND SUBTITLE Histological Evaluation of Inner Ears			5. FUNDING NUMBERS DAMD17-91-C-1120	
6. AUTHOR(S) Roger P. Hamernik, Ph.D.				
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) The Research Foundation of the State University of New York Albany, New York 12201-0009			8. PERFORMING ORGANIZATION REPORT NUMBER ARL 96-1	
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES) U.S. Army Medical Research and Materiel Command Fort Detrick, Maryland 21702-5012			10. SPONSORING/MONITORING AGENCY REPORT NUMBER	
11. SUPPLEMENTARY NOTES				
12a. DISTRIBUTION / AVAILABILITY STATEMENT Distribution authorized to U.S. Government agencies only (proprietary information, 6 Jan 97). Other requests for this document shall be referred to U.S. Army Medical Research and Materiel Command, 504 Scott Street, Fort Detrick, Maryland 21702-5012.			12b. DISTRIBUTION CODE	
13. ABSTRACT (Maximum 200)  The purpose of this contract was to support the USAARL research program on the auditory effects of blast overpressure (BOP). The statement of work for this contract identified two broad areas of responsibility; (a) to perform all the required histological work relating to the BOP projects at the USAARL and at the Albuquerque, New Mexico (EG&G) test site; (b) to assist in the on-site (USAARL) physiological and behavioral testing of experimental animals. In mid-1995, the decision was made to terminate this program (ref. Blast Program Review, Aug. 1995) and effective 30 September 1995 our on-site effort ceased, and all on-going animal experiments were terminated. The following was accomplished: 1) Atypical speaker generated impulses were shown to be suitable for modeling the effects of blast waves. 2) Correlations between audiometry and cochlear pathology were shown to be poor and that individual susceptibility to BOP trauma is highly variable. However, cochlear emissions are a sensitive, frequency specific index of BOP auditory trauma in an individual. 3) One-third octave bands of noise are suitable stimuli for audiometric testing.				
14. SUBJECT TERMS Evaluation, Ears			15. NUMBER OF PAGES 232	
			16. PRICE CODE	
17. SECURITY CLASSIFICATION OF REPORT Unclassified	18. SECURITY CLASSIFICATION OF THIS PAGE Unclassified	19. SECURITY CLASSIFICATION OF ABSTRACT Unclassified	20. LIMITATION OF ABSTRACT Limited	

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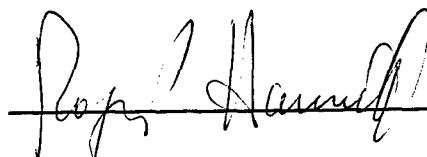
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6 Jan 97  
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Roger P. Hamernik, Ph.D.



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## HISTOLOGICAL EVALUATION OF INNER EARS

### Preface

The primary purpose of this contract was to support the USAARL Research program on the auditory effects of blast overpressure (BOP). The Statement of Work (SOW) for this contract identified two broad areas of responsibility; (a) to perform all the required histological work relating to the BOP projects at the USAARL and at the Albuquerque, New Mexico (EG&G) test site; and (b) to assist in the on-site (USAARL) physiological and behavioral testing of experimental animals. Early in the course of the BOP research it became clear from the results of published research that otoacoustic emissions could become an important tool for assessing hearing loss. Thus, an additional SOW was included in order to explore the application of otoacoustic emissions to the diagnosis of blast wave-induced hearing loss. This latter objective represents a modification to the original SOW after funds were released for the purchase of the necessary equipment. To help meet all of these objectives, The Research Foundation of The State University of New York also maintained an on-site representative (Mr. S. Hargett) at the USAARL.

In mid-1995, the decision was made to terminate the USAARL BOP program (ref. Blast Program Review, Aug. 1995) and effective 30 September 1995 our on-site effort ceased, and on-going experiments were terminated.

The following projects were undertaken and completed during the first four years of the contract:

- (a) Structural and functional changes in the auditory system of the chinchilla following exposure to high-level, speaker-generated impulses: The implication for actual BOP exposures.
- (b) Application of the cubic distortion product otoacoustic emissions to the evaluation of BOP-induced hearing loss measured using electrophysical methods (i.e. auditory evoked potentials recorded from the inferior colliculus).
- (c) A comparison of audiograms determined using one-third octave bands of noise and pure tones in the chinchilla. (Also published as USAARL Report 94-50.)
- (d) The cubic distortion product otoacoustic emissions (3DPE): A normative data base.
- (e) The prediction of PTS from a P-weighted energy model of impulse noise induced hearing loss [Note: This study was not completed.]

Following a general introduction to the nature and background of the BOP research undertaken, each of the above studies will be detailed in the body of this report. The appendices of this report contain a complete data archive for animals that have not been reported in the literature (i.e., studies (d) and (e) listed above).

## **I. Summary**

Exposure to high levels of noise in various military environments is a common occurrence and poses a serious hazard to the auditory system. In order to effectively understand the hazards and thus be in a position to develop effective standards for exposure, an interdisciplinary approach to noise hazards research must be used. An effective experimental approach (using an animal model) involves obtaining measures of auditory system performance on the unexposed subject, followed by an exposure in a calibrated sound field. Depending upon the specific nature of the physical stimulus required, the sound field can be produced by a conventional electro-acoustic sound system, or may require a more unconventional approach; for example, the use of shock tube technology or high-energy electrical discharges to produce the high-intensity transients needed to simulate BOP exposures. Following exposure the animals' audiometric variables need to be monitored at several test frequencies until they stabilize, typically after about 30-days postexposure. This procedure yields measures of temporary, compound, and permanent threshold shifts. Other measures of auditory performance such as cochlear emissions are also monitored. The audiometric data can be obtained using behavioral or physiological methods. The animals are then euthanized and the temporal bones containing the cochlea removed and preserved for anatomical study.

Quantitative evaluation of the cochlea can proceed in a variety of ways depending upon the requirements of the specific experiment. Light microscopy can be used to obtain a variety of quantitative evaluations from conventional surface preparations of the vascular and sensory epithelium. Also, cilia configurations across the organ of Corti can be assessed with scanning electron microscopy or, if still more detailed analysis is required, transmission electron microscopy can be used to assess the subcellular nature of the pathology. Each of these approaches has its range of utility and its disadvantages. Eventually, quantitative relations are developed among exposure variables, audiometric (functional) losses, and the sensory-cell pathology, which can lead to the development of experimentally-based exposure damage-risk criteria.

## **II. Introduction**

There is a consensus that the existing standards for exposure to high levels of noise are either wrong or inadequate. The basic reason for this is that there is an insufficient empirical data base upon which effective standards can be built. What is needed is a new criterion that is based upon a cohesive, systematically-acquired body of experimental data. The need for such a data base has been emphasized by, for example, von Gierke (1978, 1983), and Ward (1983), the NATO Study Group RSG.6 (1987), and the National Academy of Sciences, National Research Council (1992). One of the major objectives of the USAARL noise research program was to work toward the development of such a data base. The approach of the USAARL group was to design experiments in which animals were exposed to types of noise-exposure paradigms from which one can gain an understanding of the interrelations among the various parameters that describe a noise exposure such as the peak sound pressure level (SPL), the total energy of the exposure, the energy spectrum, temporal variables, etc., and the amount of functional change and cochlear pathology.

The task of assembling such a data base is a difficult and time-consuming process. Compounding this is the difficulty of creating, in the research laboratory, the types of traumatizing noises characteristic of BOP common in the military. Coupled to this requirement is the need to be

able to vary the parameters of a blast wave, such as peak levels, frequency spectrum, duration, etc., independently. Additionally, the experimental paradigm that is required to collect the kind of data that is useful for the development of standards requires the application of an animal model from which both audiometric and histological data can be obtained.

When trying to assess the effects of noise exposure on the auditory system the consensus is that measures of auditory function and cochlear pathology must be obtained. The reason for this is that, on an individual animal basis, the correlations between audiometry and histology are not always good. This has been demonstrated by a number of published studies, for example, Henderson et al. (1974), Hunter-Duvar and Bredberg (1974), and others. However, when large numbers of noise-damaged animals are studied the correlations are good (Hamernik et al., 1989) but variability is high. This state of affairs was the reason that measures of auditory function other than pure-tone thresholds have been sought. Considerable effort has been expended in, for example, trying to relate tuning curve measures (masked thresholds) to cochlear pathology (Davis et al., 1989). The results of this evaluation, however, were not encouraging. Although at present there is no measure other than pure-tone thresholds that is generally accepted as an adequate non-invasive measure of the effects of noise, there is increasing evidence that otoacoustic emissions may be just such a metric (Lonsbury-Martin and Martin, 1990). At this time, pure-tone threshold measures are still considered to be the basic audiometric measure used to assess the effects of noise on auditory function, and continue to be used in contemporary research on noise.

In designing experiments to study noise-induced pathology a number of compromises must be made. For example, there is a general agreement that both audiometric and quantitative histologic measures are required to properly assess the effects of noise. However, within both these realms of data, there is wide latitude in choosing the most appropriate measures. Audiometrically a decision needs to be made as to the most appropriate measure of auditory function (e.g., pure-tone thresholds, masked threshold, discrimination abilities, etc.). Similarly when evaluating the sensory structures of the cochlea, a decision must be made on which variables need to be quantified and the depth of the analysis required. As the analysis of tissue proceeds from the light-microscopic level to the electron-microscopic level, the time required to obtain data increases prohibitively, especially if large numbers of animals need to be studied thoroughly.

The effects of noise on auditory system pathology are quite variable and large numbers of animals need to be used if statistically significant results are to be obtained. Since the auditory system must be examined over a broad range of audible frequencies and anatomical variables need to be assessed over the entire continuous extent of the cochlea, approaches need to be developed which maximize the amount of quantitative data that can be obtained from each animal in a reasonable period of time so that large numbers of animals can be studied. Animal psychophysics, involving the application of behavioral-conditioning procedures to arrive at functional measures of the auditory system such as threshold estimates, is an exceedingly time-intensive process as is the process of obtaining quantitative histologic data from the cochlea. Both these tasks require specially-trained individuals that are frequently not available in a single laboratory thus further impeding the accumulation of data.

To date the method of choice in studying the effects of noise on the auditory system is to obtain measures of pure-tone thresholds at regular intervals before and after exposure. From these measures one can obtain measures of temporary threshold shifts as well as permanent threshold shifts and thus follow the course of recovery from trauma. Because of the often poor correlations between threshold data and the status of the cochlea, the condition of the sensory epithelium must be surveyed in order to quantify the anatomical pathology. Having both these realms of data available increases the confidence that the effects of the exposure have been understood. The most feasible approach for anatomically studying large numbers of animals is the cochleogram [developed by Engstrom et al. (1966)] which is a map of the distribution of sensory cells in the cochlea. Thus, the basic data pool for noise research consists of pure-tone threshold measures and the cochleogram.

An alternative to behavioral testing is the evoked electrical potential generated in the brainstem in response to a sound input. Evoked potential audiometry has the advantage that it requires less time than behavioral methods to obtain the audiogram, and is independent of animal motivation. The evoked potential system developed at the USAARL and which is in use at the SUNY ARL facility increases the efficiency with which threshold measures can be obtained.

### III. Detailed Discussion of Approach

The basic experimental protocol that was common to nearly all of the animals receiving a noise exposure consists of the following steps: (a) Preexposure measures of hearing were obtained on each animal; (b) the animal was exposed to a thoroughly documented noise; (c) following exposure, the animal's hearing thresholds or other hearing metrics were remeasured at various postexposure times; and (d) following a fixed period of recovery typically 30-days or more, the sensory structures of the cochlea were prepared for histological examination. Such a paradigm allowed for correlations to be made among variables such as (a) the physical exposure conditions, (b) temporary changes in hearing, (c) permanent changes in hearing, and (d) the extent and nature of the cochlear pathology. Variations in this basic paradigm were used, as required for a specific project, as described below. The following is a brief description of the methodology involved in those technical aspects of the research for which our (SUNY ARL) personnel were responsible.

**A. Audiometry:** All animals were prepared for audiometric testing by first surgically destroying the left cochlea (see pg. 17) and then allowing a two week recovery period. Audiometric testing was generally performed using the behavioral conditioning methods described below. Since evoked response audiometry was being developed at the USAARL, a section relating to our approach to evoked response testing is included.

1) Behavioral Training and Threshold Testing Procedures: The instrumentation and procedures for training and testing the animals were similar to those described previously by Burdick et al. (1978). Briefly, chinchillas were tested in a double-grilled cage within a 1200 Series Industrial Acoustics Company (IAC) sound room. Mounted on the cage was a row of photocells to detect the animal's location and an electronic buzzer which was used as a secondary reinforcer. A Fluke Model 6010 signal generator, an attenuator, and an amplifier were used to generate and adjust the signal level. The pure-tone signals were delivered through an Altec coaxial loudspeaker. The control, duration, and sequencing of events, as well as recording, were accomplished using a microprocessor. The behavior of the animals was monitored on a closed circuit television. The animals were conditioned to avoid an AC electric shock (1.4 mA nominal level) by crossing from one compartment to the other of a double-grilled cage during a 3.84-s trial interval during which a pulsed, pure-tone signal was presented. Each trial interval consisted of three tone pulses with 720-ms on-times separated by 560-ms off-times. The tone pulse had an exponential rise and decay function with a first time constant of 14 ms. When the avoidance response was made, the signal was immediately terminated. If the subject failed to cross from one compartment to the other during the trial interval, a shock and buzzer were presented simultaneously until the crossing response was made. This resulted in the termination of the shock, buzzer and signal.

Each group of subjects received training sessions until all subjects scored 95% correct for three successive sessions. The three sets of training sessions T<sub>1</sub>, T<sub>2</sub>, and T<sub>3</sub>, each lasted for between one and two weeks each. The three different training sessions represented increasing levels of difficulty in the listening task and response rate. During the training sessions, the animals were given one trial at each of the following nine frequencies: 0.125, 0.25, 0.5, 1.0, 1.4, 2.0, 4.0, 5.7, and 8.0 kHz. The intensities of the tones varied over a 15 dB range (50-65 dB SPL) during all the training sessions. During the first training sessions, trials were presented with an average intertrial

interval of 60 s. Then trials were presented for one or more sessions using intertrial intervals of 45, 30, and finally 20 s. Once this was accomplished, all subsequent training and testing was performed using a 20 s intertrial interval. Once the training criterion was obtained, threshold determinations were begun.

A modified method of limits (Burdick et al., 1978; Miller, 1970) was used to estimate thresholds. On the first trial of a threshold measurement, the signal level was set to 40 dB below the full output (dB) calibration level for the particular test frequency. An additional randomly-set attenuation of up to 10 dB was added to the initial 40 dB for each frequency. The initial signals could thus range from 40-65 dB SPL. A correct response at this first presentation level resulted in a further 20 dB reduction in level for the next trial and so on, until the animal failed to respond.

On the trial following a miss (failure to respond to the signal), the level of the signal was increased 10 dB and the threshold was taken as the level halfway between the lowest level that was responded to correctly and the highest level missed. After threshold values began to stabilize, which required from 8-10 complete audiograms, a threshold value was discarded if it was different from the normative values established in our laboratories by 15 dB and a second threshold measurement was taken. The threshold obtained on the second determination was always accepted. A sham trial always followed the last trial of each threshold determination. This was done to obtain an estimate of the rate of "spontaneous responding." These trials were identical to the regular trials except that the synthesizer was set to "zero" frequency and the shock and buzzer turned off. There was no consequence to the animal for spontaneous responses. Shock was turned off and only the buzzer was used as a secondary reinforcer when the signal level was within 10 dB of threshold.

Audiograms were taken until the average threshold was within plus or minus 5 dB of the average of the values established for normal animals on the five consecutive sessions. Typically, an additional five to seven audiograms were required. Then audiograms were continued until the day of exposure. The last five audiograms before exposure were averaged across sessions to produce the baseline audiogram for that particular animal. The baseline audiogram for each animal was used as a reference for computing the postexposure threshold shifts.

2) Auditory Evoked Potential (AEP): The AEP technique has been used in our laboratories (SUNY ARL) to estimate auditory thresholds for the past 20 years. Several publications (Henderson et al., 1973, 1983) detail the experimental protocol. Briefly, chronic bi-polar electrodes were implanted in the inferior colliculus for single-ended near-field recording. Under anesthesia and sterile conditions, the left bulla was entered via a posterior approach to visualize the round window. A probe inserted into the round window was used to destroy the cochlea\*. All animal surgery

\* Note: On the use of monaural animals: There is no question that destruction of one cochlea alters the efferent interactions between ears that are known to exist in binaural animals. Some recent studies have shown that efferent interactions can alter the response of the cochlea to excessive stimulation. However, the results from different laboratories are very contradictory. While species differences alone cannot account for the extent of these disagreements (i.e., Liberman, 1990 vs. Rajan, 1990). The efferent interaction studies mentioned above were performed using relatively low level, short duration, temporary threshold shift producing exposure paradigms. The experiments reported in this report are completely different; i.e., high level, PTS producing exposures where relatively large effects are to be measured. Assuming that the results of Rajan are correct, there is no good evidence that the efferent system will exert the same

conformed to veterinary standards in effect at the USAARL. A second incision was made in the midline of the skull and the periosteum retracted. A sterile bi-polar electrode was inserted under stereotaxic control into the left inferior colliculus and cemented to the adjacent bone. Electrode life was in excess of six months. The audio test signals were of 20 ms duration (5 ms rise-fall, 10 ms plateau). Signals were generated by standard laboratory audio apparatus. AEPs were collected using 100 stimulus presentations and computer averaged to obtain clear, relatively noise free responses, that is, the N1 and P1 complex between 15-40 ms following the stimulus. Threshold was estimated to be halfway between the level that generated an identifiable AEP waveform and a level that did not. The smallest test tone intensity step size was 5 dB. The estimate of threshold was made on the basis of the agreement of two out of three judgments by independent observers.

3. Cochlear Histology: Methodological Background for Anatomical Studies - General Considerations: Several traditional methods are available to study cochlear pathologies (Smith and Vernon, 1976). The method of choice depends to a large extent upon the morphological features which need to be quantified. In order to gain a perspective on the status of the sensory epithelium, a first order of analysis is often the generation of a cyto-cochleogram, which is a graphic display of the distribution of sensory cell loss as a function of distance along the basilar membrane (and hence frequency).

Typically, inner hair cell and row by row outer hair cell distributions are plotted. Such an approach represents a relatively "gross" level of analysis, but has the advantage of being able to precisely establish missing cells along the entire basilar membrane. This approach is limited to the extent that sensory cells which are present may not be functioning normally. A study of ultrastructural changes using TEM may indicate sensory cells that are present but functionally abnormal, but this approach, because of the extremely time-consuming nature of the methods, has the severe limitation of being practical to apply only to limited localized sections of the cochlea (Spoendlin, 1976). Another approach which could be used to complement the cyto-cochleogram, and which provides valuable perspectives on potentially abnormal but present sensory cells is a complete survey of stereocilia on inner and outer hair cells using SEM. The morphological integrity of cilia can be graded using a subjective rating scale of, for example, 1 to 5, where 1 represents normal appearing cilia configurations and 5 represents severely bent, fractured or missing cilia (Salvi et al., 1982; Liberman and Dodds, 1984). Other potentially useful, but generally neglected analysis are possible. (a) The quantification of the distribution of pillar cell loss (supporting cells) can be used to provide an index of nerve fiber loss. Such a loss can occur in cases in which the sensory cell populations are very close to normal (Salvi et al. 1982). (b) The quantification of VIII nerve dendrites and/or axons following sensory cell damage could yield data for correlations with psychoacoustic measures of hearing performance. (c) The quantification of the neuronal population of the complete spiral ganglion is possible but such measures are uncommon and generally not available in the literature

kind of effects in the type of exposure paradigm that we will be using. If the results of Liberman are correct then the whole issue disappears. Another point to note in regard to monauralization is derived from the experiment of Clark and Bohne (1990) in which moderate levels of low frequency noise presented on an intermittent schedule over a long time period were used. Clark and Bohne (1990) used some animals which had been surgically monauralized and others in which only the incus was removed to effect a monaural preparation. They found no difference in the results. Thus based upon the Clark and Bohne results and the contradictory findings of Liberman and Rajan, we feel that there is no compelling reason to alter our experimental protocol regarding surgical monauralization.

for pathological chinchilla cochleas. Preparation techniques to study the ganglion and obtain sensory cell populations need to be modified to include decalcification of the temporal bone as well as modification of the dissection technique when the surface-preparation approach is used. There are alternatives to sectioning the spiral ganglion in order to evaluate neural integrity in plastic-embedded cochleas, for example, sectioning of the VIII nerve axonal projections in the internal meatus. This approach should include cardiac perfusion/fixation. Alternatively using the plastic-embedded whole mount approach myelinated dendrites of the VIII nerve entering the organ of Corti through the habenular openings can be surveyed (Bohne et al. 1982). For precision and thorough evaluation of the neuronal population, both these approaches can be applied in the same animal.

All the above methods can be technically approached in a variety of ways [i.e., soft surface technique, hard surface technique (plastic embedding); with or without decalcification; fixation via cardiac perfusion and/or local *in vivo* or *in vitro* perilymphatic fixation of only the cochlea]. In addition to these preparation variables, dissection techniques need to be varied depending upon the information required. No single approach can be identified as "best" until a specific decision is made as to which class of information is required. Most of these alternate approaches to quantifying noise-induced pathologies have not been used extensively. When large numbers of animals need to be surveyed for noise-induced damage, light-microscopic analysis of surface preparation material complemented by some SEM analysis is the most commonly used approach.

Our standard approach to the analysis of the pathologic cochlea relies primarily upon the standard cochleogram. It is well accepted that the cochleogram, (i.e., a quantitative assay of the sensory cell population of the cochlea) represents, at best, a lower bound to the actual pathological condition of the cochlea. If accepted as such, the cochleogram is a valid index of the pathology and can be used in conjunction with audiometric measures to assess the effects of a noise exposure. In most situations where quantitative histology is involved there is a trade-off between the number of specimens that can be analyzed and the extent of the analysis of any individual specimen. In trying to study a phenomena in which there is a large inherent variability such as the response of the auditory system to acoustic overstimulation, the standard error can be reduced by increasing the experimental sample size. However, time limitations impose restrictions on how detailed an analysis can be performed on a single cochlea. This is the primary reason for our emphasis on the use of the traditional cochleogram. In general, a complete cilia or other detailed analysis of the sensory epithelium or cochlear nerve as a routine aspect of morphometric analysis in large numbers of animals is not performed because of the time constraints. Rather, such detailed SEM or TEM measures are usually reserved for studies where precise correlations between sensory cell function and, for example, VIII nerve physiology are required. In such studies the sample size or the region of the cochlea under study is usually very limited. The following presents the soft surface methodology used to obtain cochleograms and the SEM cochlear preparations that were used to acquire the data presented in this report. The above discussion of methods is shown schematically in the block diagram presented in Figure 1.

a) Soft Surface Histology: The standard soft surface approach to dissecting cochleas and mounting specimens for sensory cell quantification followed a well-accepted protocol. At 30 days or more postexposure the animals were anesthetized and then decapitated. Following decapitation, the two auditory bullae were removed and opened widely. The right stapes was removed and the



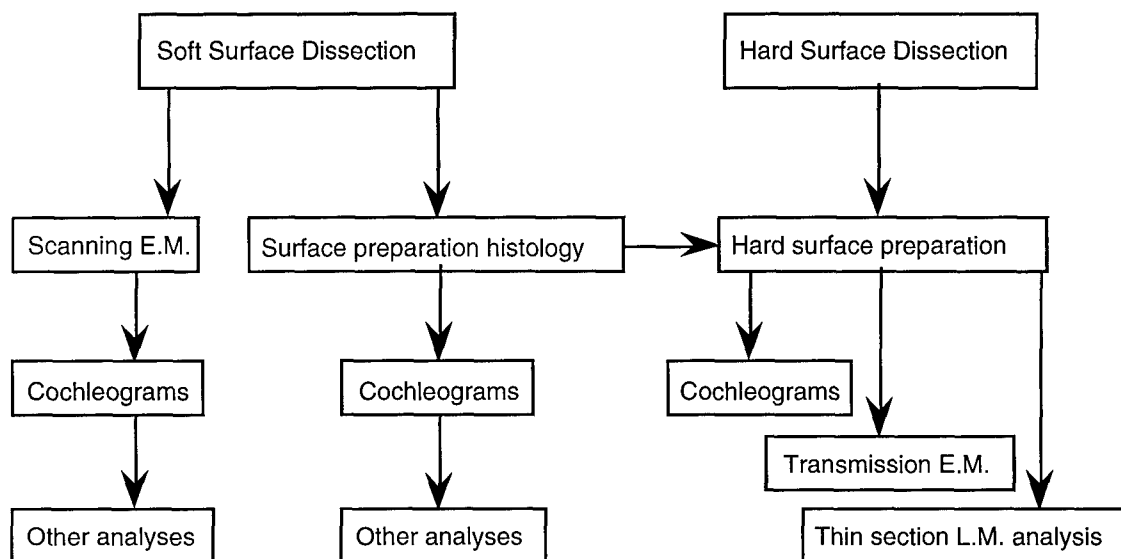


Figure 1. Schematic representation of the two basic cochlear-preparation alternatives.

round window membrane slit. A fixation solution consisting of 2.5 percent glutaraldehyde in veronal acetate buffer was then perfused through the right cochlea. Typically, the left cochlea was not perfused except for immersion in fixative since the monauralization procedure results in virtually a complete destruction of the cochlea (see pg. 17). After a variable length\* of fixation (typically on the order of 3-4 days) the right cochlea was postfixed in 1 percent osmium tetroxide in veronal acetate buffer, washed, and dehydrated to 70 percent ETOH. The entire basilar membrane and stria vascularis was piecewise dissected free from their bony attachments and mounted in glycerin on glass slides for a surface preparation, light microscopic analysis (Engstrom et al. 1966).

Inner and outer-hair cell populations were determined on a percentage basis (and in absolute terms) as a function of distance along the cochlear duct. Baseline normal sensory cell populations were established at octave lengths along the cochlea using a large population (N = 30) of normal chinchillas. Our normative population estimates are in good agreement with those published in the literature, for example, Bohne et al. (1982). Sensory cell counts which eventually yield cochleograms were performed at a magnification of 500X using a Zeiss-Nomarski light microscope. A cell was counted as missing when the cell body was not present. Alternatively, in animals that survived more than 30 days after trauma, the location of missing cells was usually well marked by a characteristic phalangeal scar at the level of the reticular lamina. Cell counts were averaged over 0.24 mm lengths of the organ of Corti as measured along a reference line established by the junction of the inner and outer pillar cells at the highest level of the reticular lamina. A frequency-place map established by Eldredge et al. (1981) was used to superimpose frequency coordinates on the length coordinate of the cochleogram so that audiometric data could be directly related to the sensory cell populations along the length of the cochlea. All the light microscopic analysis and graphics were accomplished directly using a Macintosh microcomputer system with the appropriate morphometric software developed in the SUNY ARL histology laboratory.

\* Depending upon the nature of the histological analysis required, it is often necessary to maintain a rigid schedule of fixation and staining. In such situations a lengthy and variable fixation schedual is not acceptable.

A complete presentation of data from a single animal cochlea contains the following: (a) a graph of the percentage missing inner and total outer hair cell loss as a function of distance and frequency along with that animal's permanent threshold shift, (b) printout of the actual numerical distribution of cell loss, (c) plot of each of the three rows of outer hair cell loss as in (a) above, and (d) a plot of inner and outer pillar cell loss in the format of (a) above. For each exposure group, the sensory cell losses were averaged in two ways: (a) total sensory cell loss averaged across animals in octave bands centered at 0.125 kHz through 16 kHz in eight octave steps and (b) total sensory cell losses in the entire cochlea averaged across groups. As shown in Figure 1 above, the glycerin mounted soft surface preparation could also be prepared for plastic embedding and studied with TEM or thin section LM. However, if this route was followed, the possibility of artifact is greatly increased.

b) Scanning Electron Microscopy: Cold 5% glutaraldehyde in veronal acetate buffer at pH 7.3 (630 mOsm) was gently perfused through the round window with a fine pipette. The fixed cochleas were stored overnight at 4° C. On the following day, the cochleas were post-fixed with a glutaraldehyde-osmium mixture in a 5 : 2 ratio. The glutaraldehyde was prepared as in the initial fixation and the osmium was a 2% aqueous solution. The cochleas were post-fixed for 15 min and then dehydrated with cold 35% ethanol and dissected down to the desired turn. During the dissection, the stria vascularis and spiral ligament were removed to roughly the level of the spiral prominence. The basilar membrane was left attached to the bony modiolus and the outer bony capsule. Reissner's membrane was also removed. The remaining cochlea was rapidly dehydrated in a cold-graded ethanol series (50, 70, 80, 95, 100%). The cochleas were then critical-point dried with liquid CO<sub>2</sub> following standard procedures except that no rapid pressure changes were allowed. The tissue was depressurized over a 10-15 min period. The specimens were then placed in a vacuum evaporator (Denton ◊ DV502) and the vacuum gradually increased. Gold or gold-palladium was sputtered onto the specimens using a cold sputtering head (Denton DSM-5A triode). Specimens were brought to ambient pressure using dry nitrogen and mounted onto a stub using conductive paint. The cochleas were then ready for viewing in the SEM.

## B. Database Management:

1) Data Management: All data received from the USAARL at Ft. Rucker and those collected at the SUNY ARL were entered into a comprehensive data base. The data base contains: (a) subject information (e.g., identification, group designation, etc.); (b) audiometric measurements (e.g., preexposure thresholds, recovery thresholds, and postexposure thresholds); (c) stimulus variables (e.g., total energy, octave band energies, A-weighted energies, etc.); and (d) cubic distortion product otoacoustic emissions, as required by the specific nature of the experiment.

2) Data Reduction: The data base is maintained using custom-written computer software which serves as the basis for the data appendices submitted with this report. Additional custom and commercial software packages were used to tabulate group summaries and to produce graphic presentations of the group data. Custom-written routines were used to extract data from the data base for further analysis using commercial statistical packages (e.g., SPSS release 4, SAS, etc.).

3) Statistical Analysis: The descriptive analysis of the data from these experiments consisted of: (a) a complete description of raw data and group means and standard deviations; (b) a graphical representation of all audiometric data; (c) tabular and graphical representation of individual histological summaries; and (d) group summaries of the histological analysis. Further examination of the data employed mixed model analyses of variance with repeated measures on one factor (frequency) using the SPSS statistical package.

The power of a statistical test reflects the test's ability to be used to correctly reject the null hypothesis ( $H_0$ ) and is defined as  $(1 - \beta)$ , where  $\beta$  is the probability of a Type II error. The power of a statistical test is, in general, affected by (a) sample size, (b) within groups variability, and (c) the size of the treatment effects. A number of authors provide methods to compute the power of an analysis of variance (e.g., Keppel, 1973; Hayes, 1973). In each of these descriptions, a probability value is computed from the ANOVA degrees of freedom, pooled within groups variance and expected minimum treatment effects. Thus, after an analysis is completed, the power of the ANOVA can be computed and decisions made concerning the strength of the analysis.

Since the sample size contributes directly to the calculation of the denominator degrees of freedom in the F ratio, one may estimate the sample size required to achieve a specified power. For example, in the present experiments, suppose we wished to correctly reject the null hypothesis with a probability of 0.80. A comparison of two groups of subjects with 10 subjects per group would result in one and 18 degrees of freedom for the F ratio. Therefore, if we knew the pooled within groups variance, we could define the probability that the ANOVA would detect a specified difference between the sample means, say 10 dB. If the power calculated was below 0.80, then we could increase the sample size and recompute power until the calculated probability was above the desired level. Unfortunately, this analysis requires that we know, in advance, the pooled within groups variance.

Keppel (1973) presents tables designed to assist in the determination of sample size when five other variables are specified: minimum expected treatment effects, number of treatment levels, the probability of a Type I error, the population error variance, and the desired statistical power. If we were to assume that the minimum treatment effect that had practical significance is 10 dB (twice the accuracy of our audiometric procedure) and were to estimate the population error variance to be 100 dB (based on an approximately 10 dB standard deviation from our earlier studies), with the probability of a Type I error set at 0.05, the power of a traditional t-test would be over 0.95. The power of the test given a 5 dB expected treatment effect is reduced to approximately 0.85. If we were to reduce the sample size to five subjects per group, a similar analysis would show powers of 0.85 and 0.55 for expected treatment effects of 10 and 5 dB, respectively.

Of more importance to the discussion of the power of a statistical test is the observation by Hays (1973) that the squared magnitude of the expected (or desired) treatment effects may be the most significant aspect of the calculation of the power of a statistical test. Thus, in any experiment with small treatment effects, a more powerful test must be employed to detect significant treatment effects than if the treatment effects are large. The treatment effects that we have seen in our recent studies using 10 subjects per group have exceeded 10 dB PTS in the region of the audiometric spectrum that we would expect to be most affected by the noise exposure (i.e., 4.0 kHz). Thus,

given the treatment effects seen in our earlier experiments, combined with the power analysis described above, we estimated that a sample size of between 5 to 10 subjects per group represents an appropriate compromise to assure our ability to statistically detect significant treatment effects, yet not demand an impractical amount of time and effort collecting data from excessively large numbers of subjects.

#### IV. Description and Results of Individual Studies

##### A) **Structural and Functional Changes in the Auditory System of the Chinchilla Following Exposure to High-level, Speaker-generated Impulses: The Implication for Actual BOP Exposures**

1) Summary: Immediately following exposures to various narrow- or broad-band, computer-synthesized, atypical impulses, experimental animals (chinchilla; N = 30) were euthanized and their cochleas prepared for anatomical analysis. Both light microscopic analysis of surface preparations and scanning electron microscopy were used to qualitatively evaluate the noise-induced pathologies. Results were consistent in showing severe mechanically-induced damage to the cochlear sensory epithelia. The most common form of damage was an extensive fracture of the Hensen-Deiter line of tight cell junctions at the reticular lamina. Furthermore, regardless of spectral content, the impulses produced a disruption in the junctional complex of the reticular lamina that extended over most of the extent of the organ of Corti. Exposures to atypical impulses at levels between 139 and 147 dB peak SPL were thus shown to produce essentially the same type of pathology that is produced by high-level blast-wave exposure (Hamernik et al., 1984). Such atypical synthetic impulses are suitable stimuli for modeling the effects of blast waves.

2) Background: The pressure-time waveform of noise impulses having peak SPLs of 160 dB or more is characterized by a leading edge shock wave that is responsible for producing an almost instantaneous pressure increase. These transients, common in military and some civilian environments, are the result of the rapid release of energy such as in an explosive discharge. Impulsive loading of structural elements is known to be more damaging than are steady-state loads. Exposure of an unprotected ear to such shock wave driven-transients has been shown to produce a growth of threshold shift (TS) during the first several hours after exposure (Luz and Hodge, 1971). This growth of TS has been correlated with severe mechanically-induced damage to the organ of Corti (Hamernik et al., 1988). Damage consisted of lengthy segments of the sensory epithelium being torn from their attachments on the basilar membrane as a result of the excessive membrane displacements. Structurally, the weakest area of the organ of Corti was shown by Hamernik et al. (1984) to be the line of tight cell junctions between the Hensen cells and the third row of outer hair cells and Deiter cells.

More recently, using broad-band, reverberant impacts with peak SPLs between 119 and 137 dB, Henderson et al. (1994) showed very similar patterns of mechanically-induced damage to the organ of Corti. The impulses were generated by conventional electro-acoustic methods and were meant to model industrial impacts. Their results emphasized that the severe tearing of the organ of Corti was not limited to the blast-wave type of impulses that were used by Hamernik et al. (1984), but that rather common industrial impacts could also produce the same type of an acoustic trauma. Early studies by Davis et al. (1953) and Spoendlin (1976) using intense steady-state noise showed that such mechanically-induced, immediate postexposure damage was furthermore not limited to just impulsive noises. An important practical and public health difference, however, is that unprotected exposures to high-level impacts are relatively common while unprotected exposures to steady state noises of 130 dB or more are not.

It is generally accepted that the hazard to hearing from exposure to noise transients whether impulse, impact, or a synthetic waveform is dependent upon peak levels, total energy, spectrum and temporal variables of the exposure. In addition to stimulus variables, the nature of the response of the cochlea to a specific stimulus is considered to be important in predicting the hazards of exposure. For example, there has been a long-standing idea of a critical level, that is, the level above which hearing loss from impulse-noise exposure grows very rapidly. This critical level has also been identified as the level above which the mechanism of damage in the cochlea shifts from metabolic to mechanical. It is clear that once the mechanism of damage shifts to the mechanical, the sensory epithelium rapidly deteriorates.

The development of criteria for exposure to impulse noise relies on animal model experiments to understand the effects that various parameters of the stimulus have on auditory function. Such studies have contributed to developing, for example, spectral-energy weighting functions, time-intensity, and intensity-number trading relations. Such relations are important in the development of military or civilian exposure criteria. In order to understand the effect that a specific parameter has on hearing function in a controlled experimental situation it is often necessary to keep the

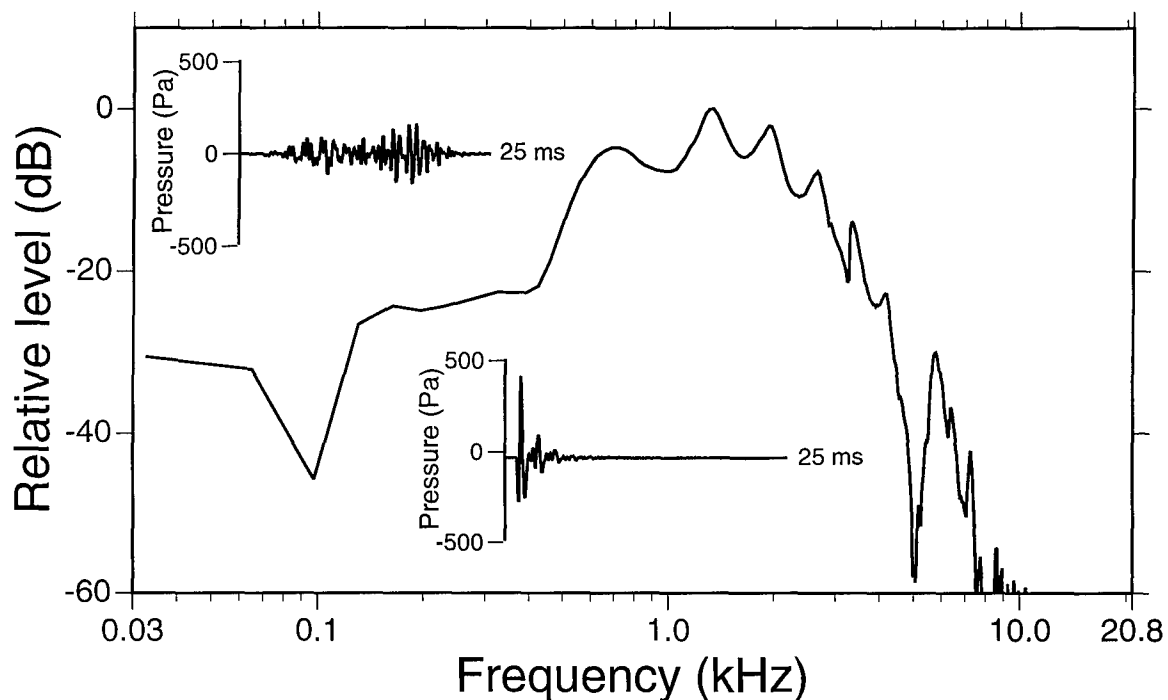


Figure 2. The pressure-time waveforms and common spectrum of the 139 and 147 dB peak SPL broad-band impacts.

remaining parameters constant. Achieving this is not always possible with naturally occurring impulses, and recourse must be made to very atypical impulses, that is, synthetic impulses designed with specific parametric characteristics. For example, the spectrum of an impulse has a strong effect on hearing loss, (Price, 1979, 1983, and 1986; Hamernik et al. 1991). Thus, the relation between peak pressure and the total energy can be determined only if the spectrum of the impulse and remaining exposure variables are fixed (Patterson et al., 1986; Patterson, 1991). Similarly,

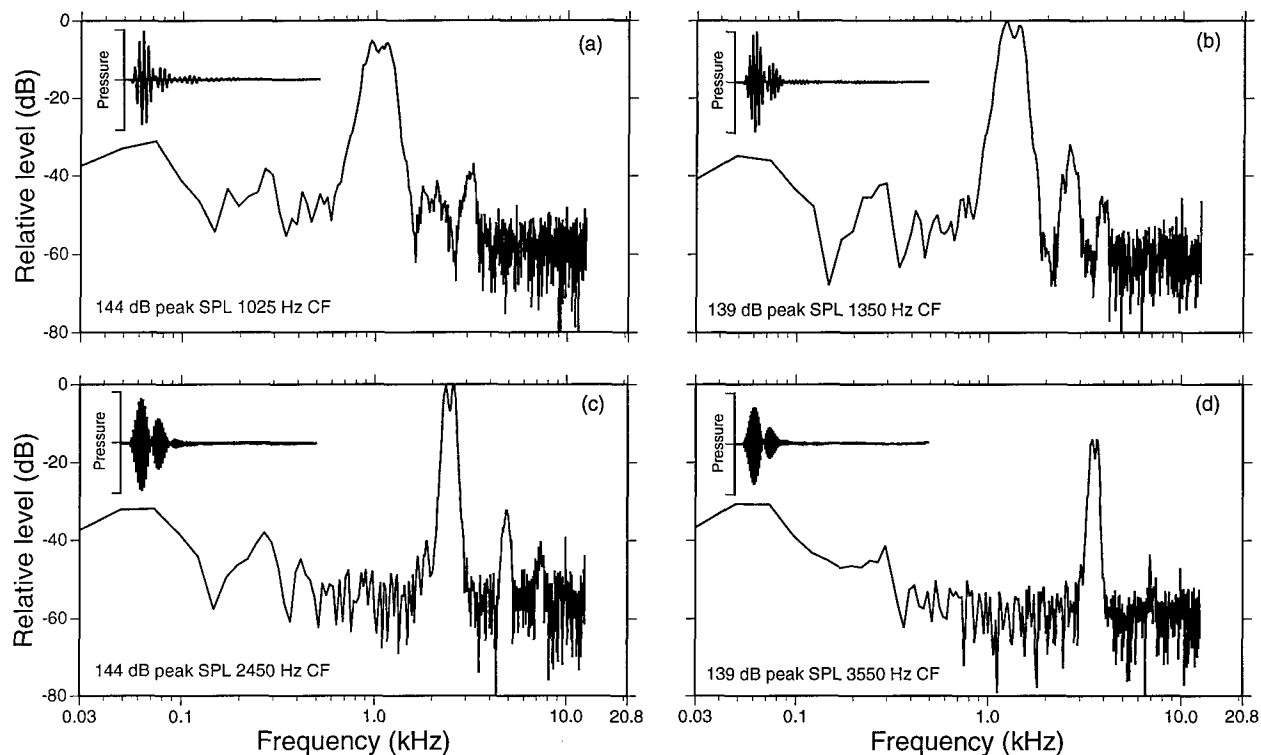








Figure 3. The pressure-time waveforms and spectra of the four different narrow-band impacts.

narrow-band impulses have been used as stimuli in the development of weighting functions (Patterson et al., 1993). The pressure-time histories of these synthesized impulses are very different from those that occur in industrial or military environments. For the results of studies that use such atypical or “designer” impulses to be applicable to the development of standards for exposure to real-life blast waves that can exceed 185 dB peak SPL, it is important that the effects these synthetic impulses have on the cochlea be similar to the effects produced by the actual blast waves.

The purpose of this study was to show that the anatomical correlates of exposure to atypical synthetic impulses are similar to the effects of high-level blast waves, and that these synthetic waveforms are, therefore, suitable stimuli for studying the effects that realistic blast waves have on the cochlea.

3) Methods and Procedures: Anatomical data, using either light microscopic analysis of surface preparations or scanning electron microscopy (SEM), were collected on 30 chinchillas exposed to one of six different synthetic impulses. Two of the impulses were broad-band stimuli having peak SPLs of 147 and 139 dB. The spectra of these two impulses were the same but their temporal structures, shown in Figure 2, were very different. Both impulses had the same energy spectrum, and the total energy in each of the exposures was the same. The other four impulses were narrow-band impulses approximately 400 Hz wide, independent of center frequency (CF). The following CFs and peak SPLs were used: 1025 Hz/144 dB; 1350 Hz/139 dB; 2450 Hz/144 dB and 3550 Hz/139 dB. The pressure-time waveforms and spectra of these impulses are shown in Figure 3. All the above exposures consisted of 100 impulse presentations; 1/3s. The methods used to generate these impulses can be found in Patterson et al. (1986) and (1993).

Table 1. Damage key used in the evaluation of impulse noise-induced damage in individual chinchilla cochleas.

	Disrupted hair cells or cilia.		Severe crack in the outer hair cell (Dieter cell) / Hensen cell junction. The mass of Hensen cells may have separated from the reticular lamina.
	Irregular Hensen cell / Dieter cell junction.		Full separation of the Hensen cells at the outer hair cell border. Hensen cells missing or torn off the basilar membrane.
	Separation of the outer hair cell (Dieter cell) / Hensen cell junction.		Large fracture within the organ of Corti which may include separation of the sensory epithelium from the basilar membrane (i.e., between outer pillar cell border and the first row of outer hair cells, or between the second and third rows of outer hair cells).

Animals were restrained and individually exposed with the external canal oriented at a normal angle of incidence to the sound source. Immediately following exposure, the animals were anesthetized with an isoflurane and nitrous oxide inhalation and euthanized by decapitation. Each bulla was quickly removed and the cochlea perfused with cold 5% glutaraldehyde in veronal acetate buffer at pH 7.3. After primary fixation, the cochleas were perfused with cold 1% osmium tetroxide in veronal acetate buffer at pH 7.3 for 30 minutes. At this point, some of the cochleas were prepared either for SEM or surface preparation (Engstrom et al., 1966) viewing. The cochleas randomly chosen for surface preparations were dehydrated to 70% ethanol in a graded series, and each organ of Corti was completely dissected and mounted for light-microscopic viewing. Based upon the surface preparation, the approximate location of any lesions were noted under the light microscope and used as a subsequent guide to the preparation of the remaining cochleas for SEM viewing. The cochleas that were prepared for SEM were first dissected to a predetermined location and the removed tissue mounted as a surface preparation. The remainder of the cochlea was then further prepared for SEM. Each SEM specimen was dehydrated to 100% ethanol in a graded series and critical point dried in a Denton unit using CO<sub>2</sub>. Following drying, the cochleas were mounted on SEM stubs with low-resistance contact cement and gold was sputtered onto each specimen with a Balzer SCD-004 sputter coater. The cochleas were viewed using a Zeiss DSM 940 scanning electron microscope operating at 10-30 KeV. Polaroid photomicrographs were taken from the screen. The light-microscopic analysis was performed with a Zeiss Universal equipped with Nomarski optics.

Each of the 30 cochleas was evaluated subjectively using a six-point scale reflecting the nature of the damage as described in Table 1. This evaluation was performed without prior knowledge of the exposure condition. In many regions of the noise-damaged cochleas, more than one damage code might apply; thus, in the graphical presentation of the pathology in each cochlea, more than one symbol was often used. The cochlear frequency map used in the graphical presentation of data was that reported by Eldredge et al. (1981).



4) Results: The group mean permanent (30-day postexposure) audiometric and histological effects of these exposures are shown in Figure 4. These data, taken from Patterson et al. (1986 and 1993), serve as a reference for evaluating the immediate postexposure morphology resulting from exposure to the same series of exposures. The group mean permanent threshold shifts (PTS) for these exposures is typically 40 dB or more and the losses are distributed relatively uniformly across a broad range of audiometric test frequencies. Sensory cell losses consist primarily of outer hair cell (OHC) losses which are relatively similar across the various exposure groups except for the 2450 Hz CF group which showed the least amount of OHC loss. Inner hair cell losses were much smaller and more variable across the exposure groups.

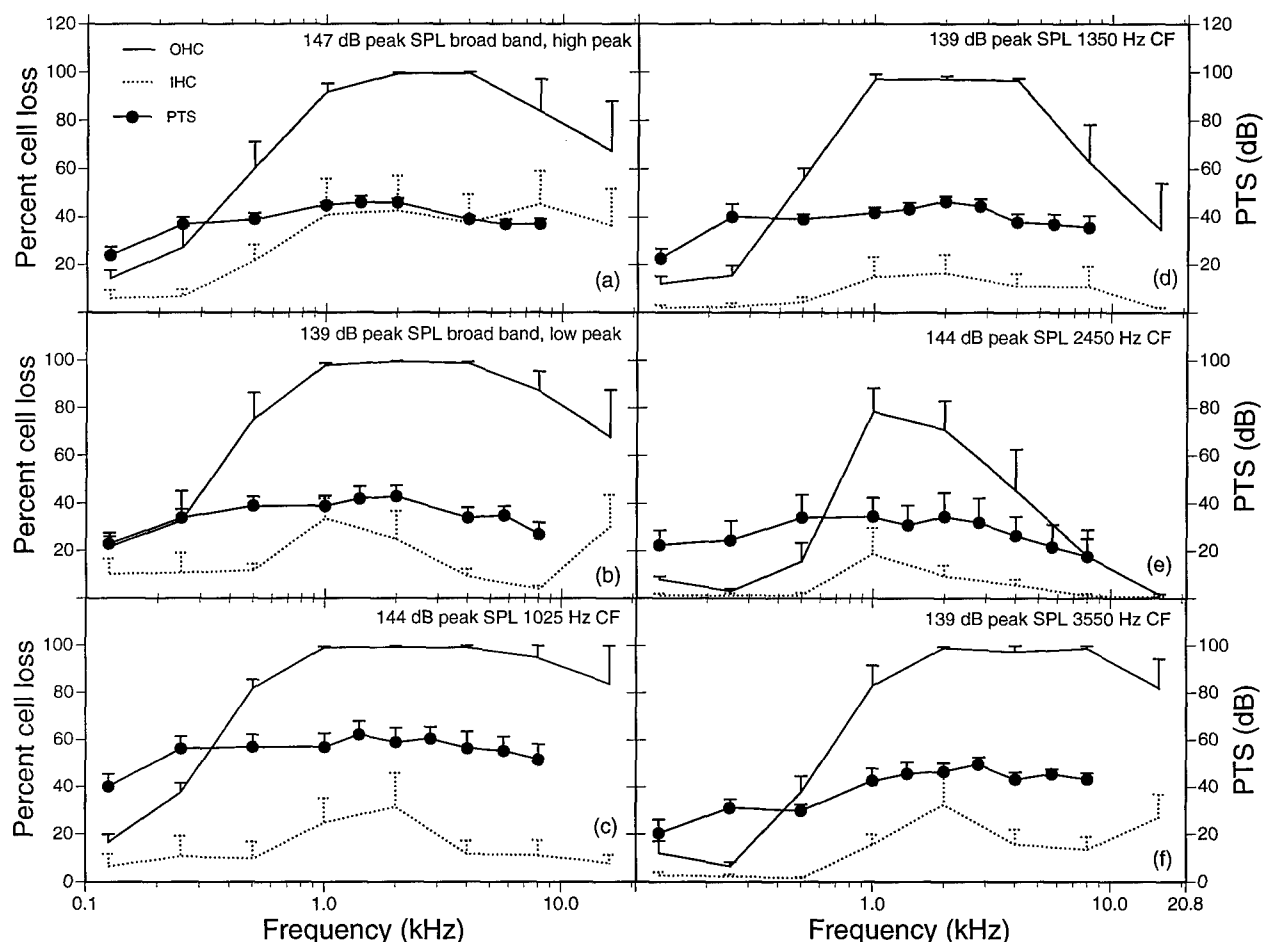


Figure 4. The group mean permanent threshold shift (PTS) audiograms and cochleograms for the exposure condition identified in each panel. Bar indicates one standard error of the mean.

Figure 5 shows the results of the subjective evaluation of each cochlea immediately following the exposure. In regions of the cochlea where no damage symbol is shown the organ of Corti appeared normal. The regions labeled "no observations" indicate areas that could not be evaluated because of limitations of the SEM and dissection methods that were used. All the cochleas examined showed fractures of the tight cell junctions most frequently in the Hensen cell/OHC (Deiter cell) junction at the level of the reticular lamina. This damage frequently extended throughout most of the cochlea in those specimens that were completely analyzed. A disruption of the integrity of the

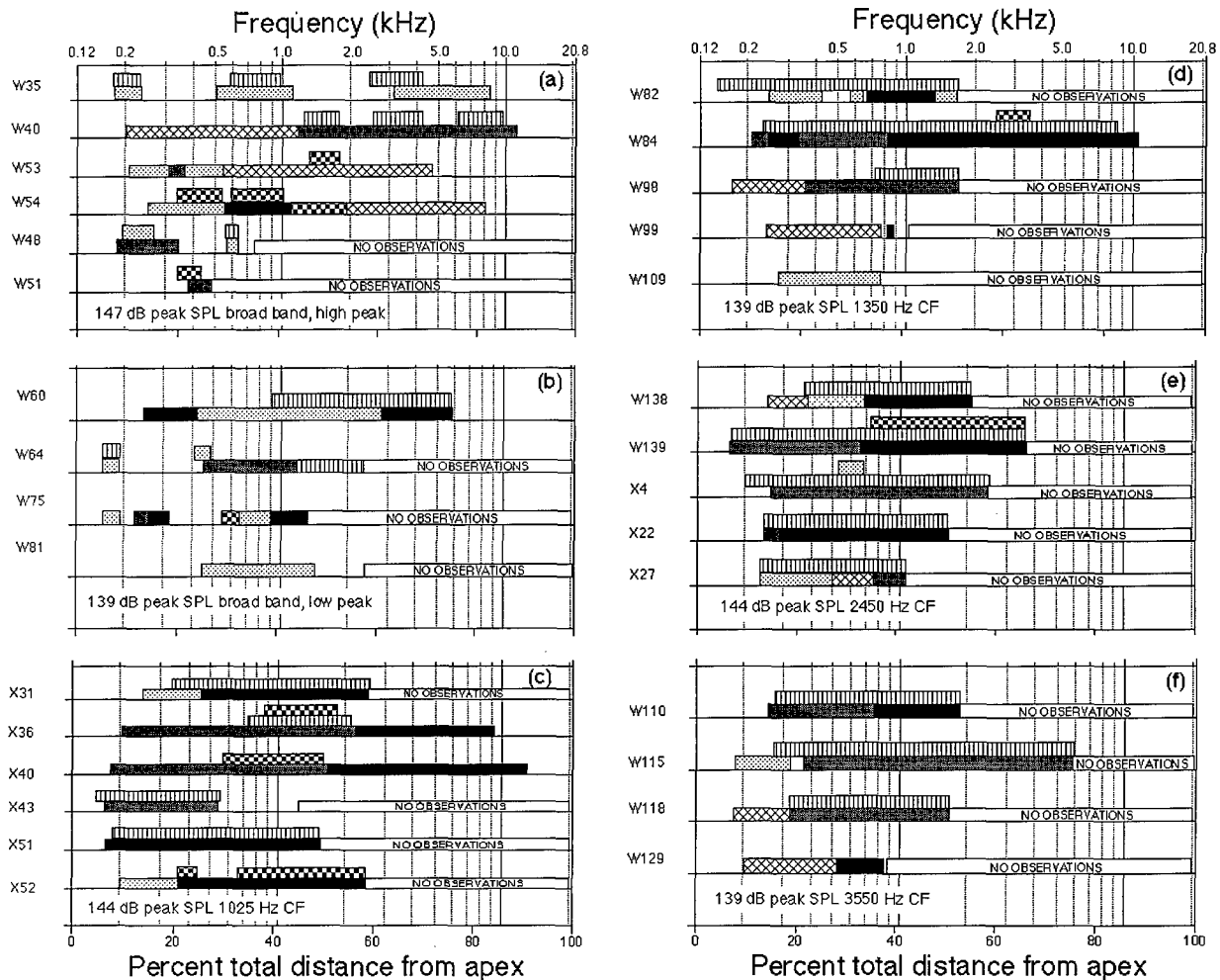


Figure 5. A graphical presentation of the pattern and nature of the impulse noise-induced damage in each cochlea of the six experimental groups identified in each panel. The damage key is explained in Table 1.

tight cell junctions on the reticular lamina and the subsequent intermixing of intracochlear fluids have been implicated by Bohne and Rabbitt (1983) in the continuing postexposure deterioration of the sensory cells. The apical 10 to 15% of the cochlea, regardless of the impulse CF, always showed some evidence of fracture of the tight cell junctions. In general, the subjective evaluations reflect the final status of the sensory epithelia as seen in the group mean 30-day postexposure cochleograms shown in Figure 4.

The series of micrographs shown in Figures 6 through 9 show the detailed nature of the damage in selected exposure groups. Since the basic type of damage was surprisingly similar for each exposure condition, regardless of the impulse used, only a sampling of the pathologies is presented. The greatest variation within a group as well as across exposure groups is in the severity of the damage rather than in the nature or location of the damage. The interesting as well as significant feature of these results is that, regardless of the spectrum of these impulses, the damage pattern extends from the apical regions of the cochlea to within about 15% of the distance from the basal end of the cochlea in many animals. This is true even for the 400 Hz-wide narrow-band impact with a CF at 3550 Hz. For this impact, maximum displacements on the basilar membrane

should be localized to the basal 30% of the basilar membrane. The following micrographs summarize the morphological findings for the indicated exposure conditions.

a) 2450 Hz CF exposure at 144 dB peak SPL: Figure 6 shows, in a low magnification SEM, the appearance of the organ of Corti in a region of a severe fracture and dislocation of the sensory epithelia. This structural failure is in the 0.4 to 0.6 kHz region of the cochlea and extends towards the base, beyond the 4.0 kHz location. Immediately noticeable is the long expanse of the sensory epithelia (S) that has been torn from its attachments on the basilar membrane. The tectorial membrane (TM) is seen rolled back toward the modiolous revealing the marginal net specialization (arrowhead). Also visible is some damage to the TM in the form of a tear in the marginal net (double arrowheads). The mass of Hensen cells (H) has been separated from the basilar membrane as well as from the third row of OHCs. The Hensen cells have been dislodged from their attachments over a longer distance than have the sensory cells, suggesting that the Hensen cells were the first to have been affected by the noise exposure. The inset shows the appearance of this cochlea as seen through the dissecting microscope just after the bony shell and a portion of the upper turn were removed. The fracture and dislocation are seen to extend in excess of a full cochlear turn. The nature of this mechanical damage is very similar to that shown in Figure 4 of the Hamenik et al. (1984) paper, which was produced by a high-level blast-wave exposure. Panels C and D show the 1.0 and 2.0 kHz regions of the organ of Corti in two other animals (X27 and X4). The fracture ridge between the Hensen cells and the third row of OHCs can be seen (arrowheads) along with some exuding of cellular material. A similar but more severe fracture is seen in the 0.8 kHz region shown in Panels E and F along with the extrusion of substantially more cellular material. The cilia on most of the OHCs are also severely damaged or missing. The above description is the general finding across all the various exposures. Only the severity and extent of the damage varied.

b) 1350Hz CF exposure at 139 dB peak SPL: Figure 7A shows a surface preparation of the 1.5 kHz region from Chinchilla W84. The region between the arrowheads in the low magnification inset shows the extent of the Hensen cell separation shown in Panel A. Panel B shows a similar fracture in the 0.2 kHz region of Chinchilla W82. In this example, the OHC cilia are severely damaged and the IHC cilia appear slightly disordered. In Panel C, showing the 0.8 kHz region in Chinchilla W98, the IHCs and OHCs and their cilia appear in good condition despite severe separation (arrow) of the Hensen cells.

c) 1025 Hz CF exposure at 144 dB peak SPL: In Figure 8 Panels A and D, the sensory epithelia is shown just apical to the region where the organ of Corti is torn loose from the basilar membrane in Chinchilla X51. Panel D shows the Hensen cells completely separated from the OHCs (double arrowheads) and another less commonly observed fracture between the second and third rows of OHCs (double arrow). Panels B and C show similar regions in Chinchilla X31. In Panel C the Hensen cells are gone and the phalangeal processes (p) of the Deiters cells are visible.

d) High peak (147 dB) and low peak (139 dB) broad-band impact exposures: Essentially identical pathologies were observed in the broad-band impact exposures. A surface preparation example is shown in Figure 9. Long segments of the Hensen cell strip have been torn loose. The fracture extends from the apical turn as far as the 10.0 kHz region in some cochleas. In some cases the fractures are very localized but the fracture region often reappears farther towards the base with relatively normal-appearing sensory epithelia between these areas.

Legend to Figure on Page 32

*Figure 6. Exposure: 2450 Hz CF at 144 dB. A series of SEM micrographs from various chinchillas (W139, X27, and X4) showing in Panels A and B the severe separation of the sensory epithelia (S) and the Hensen cells (H) from the basilar membrane immediately following exposure. The remaining panels show at higher resolution the fracture ridge along the line of attachment of the Hensen cells at the reticular lamina (arrowheads). TM-tectorial membrane, e-extruded cellular material, IHC-inner hair cells, OHC,1,2,3,-three rows of outer hair cell.*

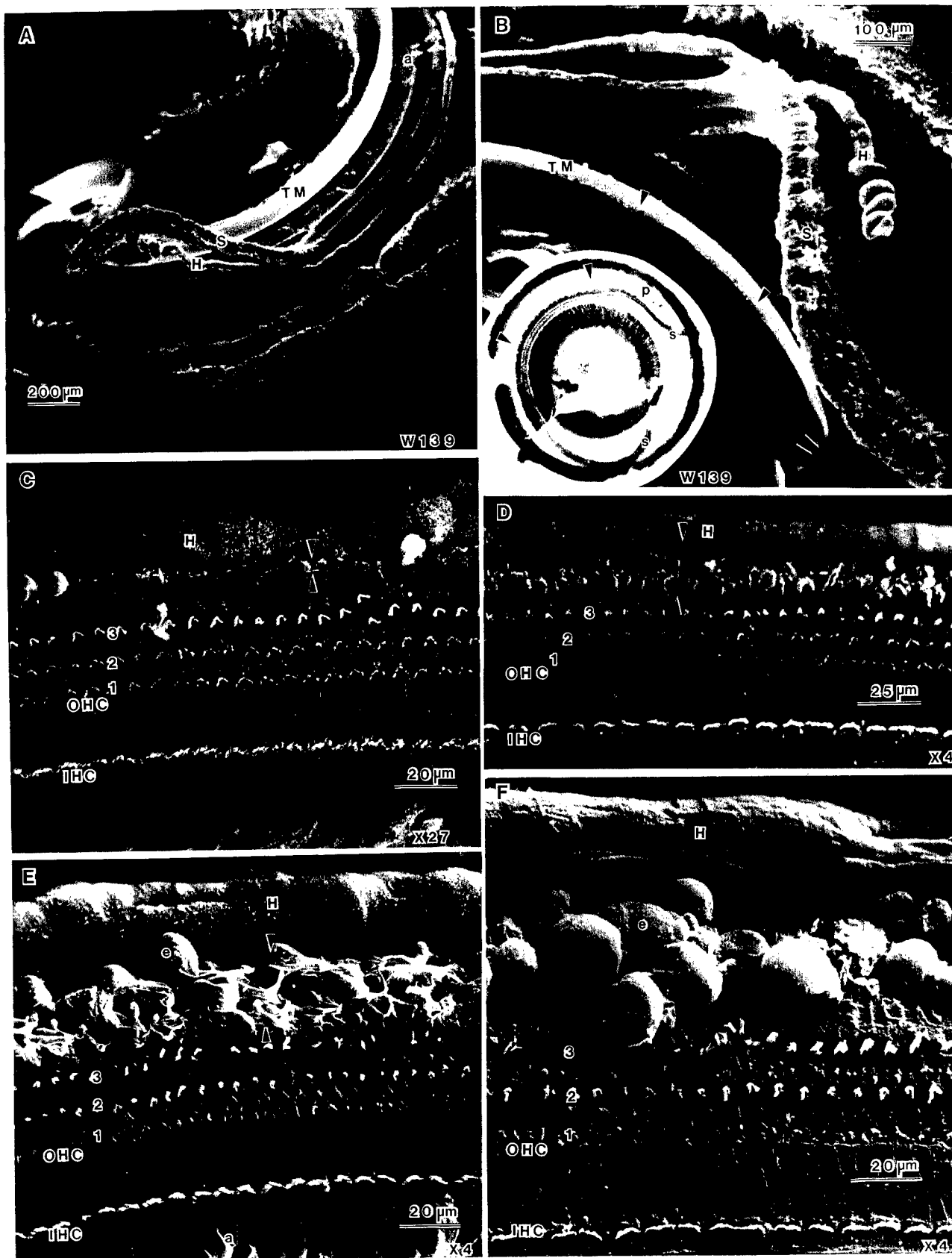


Figure 6

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*Figure 7. Exposure: 1350 Hz CF at 139 dB. Panel A shows a surface preparation of the organ of Corti from the 1.5 kHz region of Chinchilla W84. The mass of Hensen cells is clearly separated but still adjacent to the sensory epithelia. The low magnification inset of the same specimen shows (arrowheads) the considerable extent of this fracture ridge. Panels B and C show the 0.2 and 0.8 kHz region of Chinchillas W82 and W98, respectively. In Panel B, the Hensen cell fracture ridge (arrowheads) is associated with severely damaged outer hair cells (OHC) and disrupted inner hair cell (IHC) cilia while in Panel C the severe Hensen cell fracture is associated with a region of relatively normal-looking sensory cells.*



Figure 7

Legend to Figure on Page 36

*Figure 8. Exposure: 1025 Hz CF at 144 dB. The 0.2 (Panel A) and 0.5 kHz (Panel D) region of Chinchilla X51 is shown. In Panel A, the Hensen cells are separated and the outer hair cells damaged. In Panel D this separation is complete (double arrowheads) and a second fracture is seen between the second and third rows of outer hair cells. In Panel B and C, from the 0.2 and 0.5 kHz region, respectively, of Chinchilla X31, the separation of the Hensen cells develops within a short distance into a complete tearing loose of the entire cell mass (Panel C) exposing the phalangeal processes (p) of the Deiter cell.*



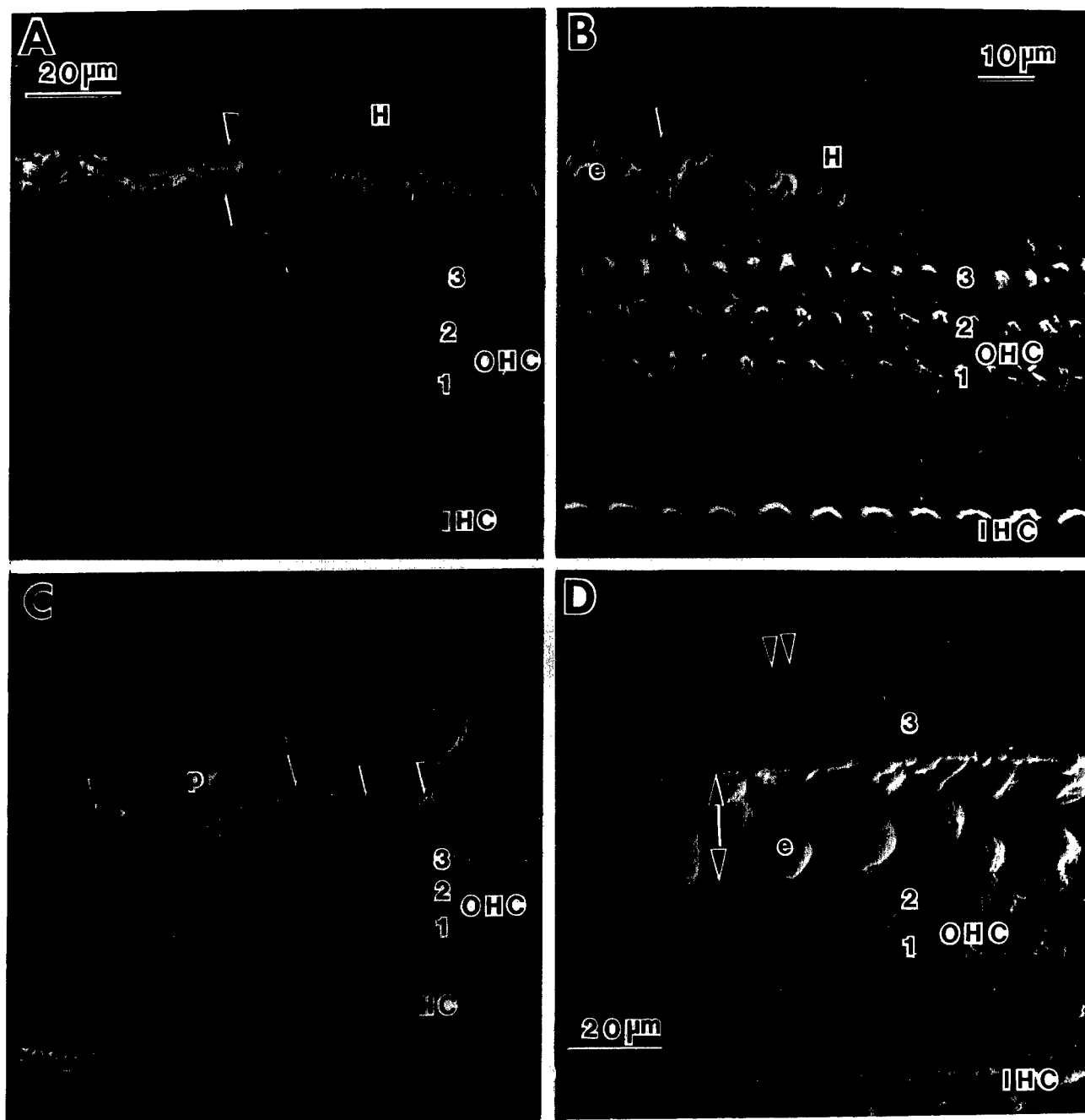


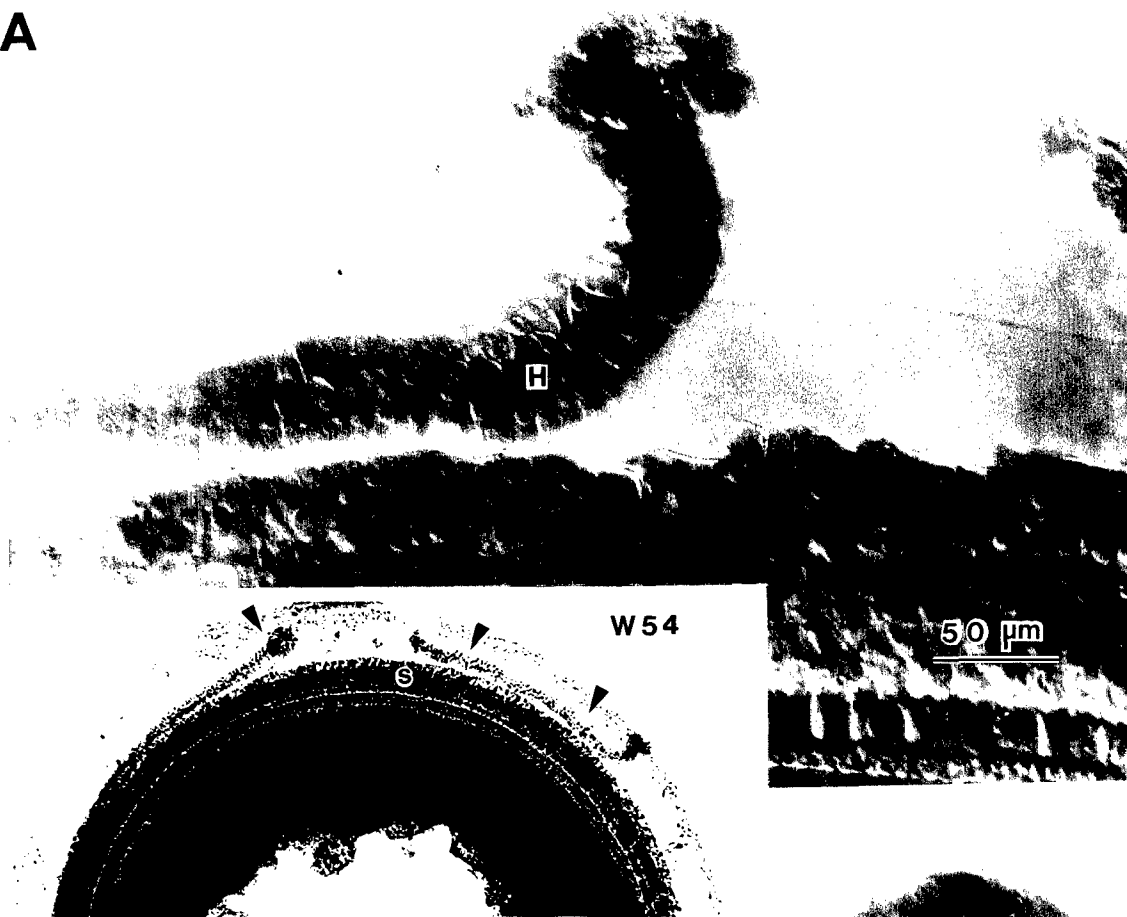
Figure 8

5) Conclusions: Despite the atypical physical characteristics of the synthesized impulses used in these exposures, the pathologies across the various groups were surprisingly similar. The nature of the damage was also very similar to that reported by Hamernik et al. (1984) following high-level blast-wave exposure. Thus, the use of atypical synthesized impacts in the development of relations among exposure parameters for use in the development of exposure criteria is justified.

Legend to Figure on Page 39

*Figure 9. Exposure: 147 dB broad-band impact. Surface preparations showing the wide spread loss and separation of the Hensen cells from the 0.5 kHz region of Chinchilla W54 (inset). Panel A and B show higher resolution micrographs of the basal and apical ends of the separated Hensen cell mass.*

A



B



Figure 9

**B. Application of the Cubic Distortion Product Otoacoustic Emissions to the Evaluation of BOP-induced Hearing Loss Measured Using Electrophysical Methods (i.e., Auditory Evoked Potentials Recorded From the Inferior Colliculus)**

1) Summary: A normative study of the cubic distortion product emissions from 104 monaural and binaural chinchillas was undertaken to establish criteria upon which noise exposed animals could be evaluated. From this normative group, 47 randomly-selected chinchillas were exposed to various high-level (150, 155 and 160 dB peak SPL) impulse noises. AEP and cubic distortion product otoacoustic emissions (3DPE) were measured on each animal pre- and postexposure and related to the sensory cell populations 30 days postexposure. Both group mean and individual animal data indicated that the distortion product emissions were more sensitive, frequency-specific indices of noise-induced cochlear effects than pure-tone threshold measures. This was particularly evident near the threshold for noise-induced damage to the outer hair cell system.

2) Introduction: From the time that cochlear emissions were shown to be a consistent product of normal cochlear function (Kemp, 1978) there was an optimism that emissions could be developed into a sensitive, noninvasive diagnostic tool (Kemp, 1988; Lonsbury-Martin and Martin, 1990). Since the source of the emissions lies in the bi-directional transduction properties of the outer hair cells (OHC) (Brownell et al., 1985; Brownell, 1990), emissions seemed ideally suited for the early detection of noise-induced hearing loss (NIHL) which typically first manifests itself in OHC changes and, in particular, the OHC cilia (Liberman and Dodds, 1984).

In animal models of NIHL the 3DPE has been shown to track the temporal and frequency pattern of threshold shift (Schmiedt, 1986; Franklin et al., 1991). While the correspondence between the dynamics of the 3DPE and threshold shifts (TS) was confirmed by Subramaniam et al. (1994), the correlation between the extent of OHC changes and the 3DPE was not good. Considerable OHC loss and cilia disarray was found despite normal 3DPEs. Inconsistent relations among 3DPEs, threshold measures, and sensory cell pathology have been reported from widely different experiments (e.g. Canlon et al., 1993; Subramaniam et al., 1995). The correlations between sensory cell populations and pure-tone thresholds in individual animals are similarly not always good (Hamernik et al., 1989). Part of this difficulty can be traced to the surface preparation technique (Engstrom et al., 1966) which is commonly used to quickly and accurately quantify the sensory cell population of the cochlea in a frequency/place-specific manner. Its main drawback is its inability, at the light-microscopic level, to easily quantify other morphological changes, such as cilia defects, that may affect the function of cells that are present. Anatomical methods that are used to quantify the more subtle changes are extremely time consuming, especially if the entire cochlea is to be surveyed. Cochlear emissions may obviate some of these difficulties if good correlations between the emissions and cell pathology can be established (Brown et al., 1989).

This paper presents some of the results of a large sample (N=104) normative study of the 3DPE in the chinchilla along with the relations among group mean and individual measures of threshold shift, sensory cell loss, and 3DPEs from 47 animals that received an acute exposure to various high-level reverberant noise impulses.

### 3) Method

A) Subjects: The chinchilla served as the experimental animal. The normative data base was established from 104 monaural animals. Animals were made monaural, as part of the evoked response audiometry protocol, by the surgical destruction of the left cochlea. Nine of these animals were also tested in the right ear for 3DPEs prior to monauralizing surgery. Forty-seven of the monaural animals, randomly selected, were used in the acute impulse noise exposures. All postexposure changes in emissions were averaged within and across animals within each exposure group and compared to their respective preexposure values to establish the effects of the noise exposure.

B) Noise Exposure: Impulses were produced by a compressed air-driven shock tube. Each animal was individually exposed to either 150, 155, or 160 dB peak SPL transients; 1, 10, or 100 times. The interval between multiple transients was one minute and all exposures took place in a hard-wall enclosure in order to produce a reverberant waveform. The number of animals in each exposure condition was variable and is indicated in the graphical presentation of the results. A pressure-time history and spectrum of the 155 dB reverberant blast wave is shown in Figure 10. The 150 and 160 dB impulses essentially differed only in the temporal and spectral amplitudes. The A-weighted energies of these impulses peaked in the 1.5 to 4 kHz region (Ahroon et al., 1995).

C) Audiometry: Preexposure hearing thresholds were estimated on each animal using the auditory evoked potential (AEP) recorded from an electrode implanted in the inferior colliculus. Additional details of the AEP procedures can be found in Henderson et al. (1973) and Ahroon et al.

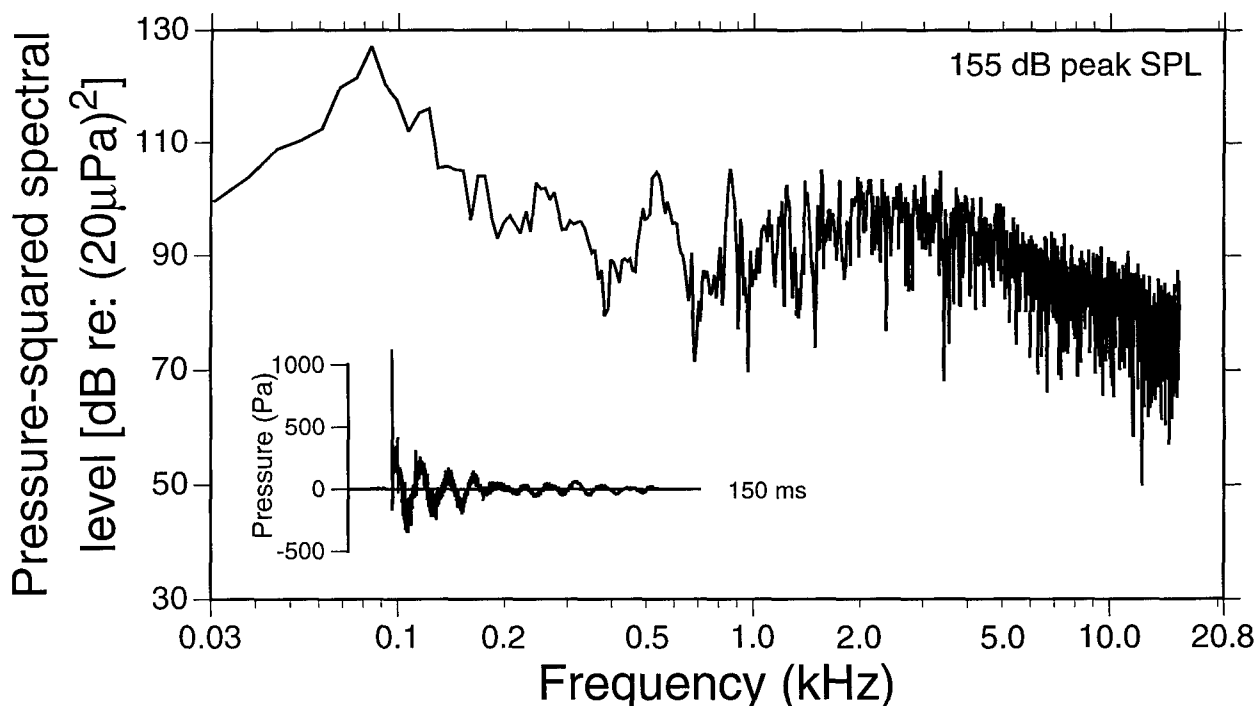


Figure 10. The spectrum and the time waveform of the 155 dB peak SPL impulse produced by the shock tube. The measurement was made in a hard-walled enclosure at the position of the animal's external meatus.

(1993). Thresholds were measured at octave intervals from 0.5 kHz to 16 kHz and at the half-octave frequency of 11.2 kHz. The average of three separate threshold determinations at each frequency obtained on different days was used to establish each animal's audiogram. This same protocol was followed on each animal 30 days after exposure and the difference between the two audiograms was accepted as that animal's permanent threshold shift (PTS). Middle ear function was checked in each noise-exposed animal before and after exposure using a Grason-Stadler 1723 middle ear analyzer. All noise-exposed animals showed normal pre- and postexposure tympanograms at 220 and 660 Hz (Eames et al., 1975).

D) Cubic Distortion Product Otoacoustic Emissions (3DPE): The Virtual Model 330 Otoacoustic Emissions Test Instrument was used to collect 3DPE data. Data collection consisted of two parts. (a) The establishment of a normative data base consisting of 102 preexposure (monaural) animals from which 3DPE data were acquired as a function of frequency (DPEgrams) at two primary intensities and 104 animals from which 3DPE input/output (I/O) functions [i.e., 3DPEs as a function of primary level at a given frequency (Schmiedt, 1984)] were acquired at six frequencies. (b) The collection of DPEgrams and I/O functions from 47 animals exposed to various blast waves. In addition, a subset ( $N = 9$ ) of the animals in (a) above were tested in the right ear with the Virtual 330 prior to surgery (i.e., while still binaural) and the results compared to the mean data obtained from the monaural animals. These nine binaural animals were tested three times daily for 25 days to examine test-retest reliability. Following monauralization and a two-week recovery period, these animals were again tested three times/day for an additional five days.

3DPE Experimental Protocol: During all 3DPE testing, the animals were awake and restrained. The 3DPE collection parameters were: eight time averages;  $f_2/f_1 = 1.22$ , where  $f_1$  and  $f_2$  are the lower and upper primary tone frequencies respectively; DPEgrams measured using 55 and 65 dB SPL primary tones with  $L_1 = L_2$  ( $L$  = primary tone level); 3DPE plotted as a function of  $f = (f_1 f_2)^{-0.5}$ , where  $500 < (f_1 f_2)^{-0.5} < 8000$  Hz, at 1/6 octave steps; I/O functions obtained in 5 dB steps at  $(f_1 f_2)^{-0.5} = 1, 2, 3, 4, 6$ , and 8 kHz. Three sets of DPEgrams and I/O functions were obtained on different days on each animal and the mean used to establish that animal's preexposure 3DPEs.

3DPEs Following impulse noise exposure: Forty-seven (47) animals, which were a subset of the normative study, were exposed to various blast waves. Thirty days following exposure, three DPEgrams using 65 and 55 dB primaries and three sets of I/O functions were recorded on three different days. The mean for each animal was used as the 30-day postexposure DPEgram or I/O function for comparison with that animal's preexposure data.

E) Cochlear Histology: Following postexposure audiometric and emission testing, the animals were euthanized under anesthesia by decapitation and the cochleas immediately removed and fixed. The cochleas were dissected and the status of the sensory cell population evaluated using conventional surface preparation histology (Engstrom et al., 1966). Briefly, the stapes was removed and the round window membrane opened to allow transcochlear perfusion, via the scala tympani/scala vestibuli with cold 2.5% glutaraldehyde in veronal acetate buffer at 7.3 pH (605 mOsm). Postfixation was performed on the following day with one percent osmium tetroxide in veronal acetate buffer (pH 7.3) for 30 minutes. The cochleas were then dissected and the entire sensory epithelium was mounted in glycerin on glass slides. The status of sensory and supporting cells were evaluated with Nomarski differential interference contrast microscopy and entered into

a data base on a laboratory Macintosh computer. Standard cochleograms were then constructed by computing the percent sensory cell loss across the length of the cochlea in 0.24 mm steps. These cell-loss figures were then converted into percent loss over octave bands centered at the audiometric test frequencies along the length of the cochlea and correlated with the frequency-place map of the chinchilla cochlea constructed by Eldredge et al. (1981). No evaluation of the cilia was made.

4) **Results.** The mean DPEgram based on 102 monaural (left cochlea destroyed) chinchillas and the mean I/O functions based on 104 monaural chinchillas are shown in Figures 11 and 12 respectively. The mean 3DPE data for  $f = (f_1 f_2)^{-0.5} < 1.0$  kHz are not distinguishable from the noise floor for primary levels less than about 55 dB SPL. Above 1.0 kHz the 3DPE is robust and the noise floor rapidly decreases. In subsequent figures, only emission data points that lie above the noise floor are shown. The mean I/O functions shown in Figure 12 are similar to those reported by Subramanian et al. (1994) except for the 1.0 kHz function where our noise floor is considerably (20 dB) higher. Thresholds at 1.0 kHz for the Subramanian et al. data are about 35 dB SPL. The filled symbols in Figure 11 represent the 3DPE data for the 55 and 65 dB primaries taken from the I/O functions. The DPEgram and I/O data acquisition protocols yield consistent results.

When the 3DPEs were collected on the (N=9) sample of normal binaural animals there was virtually no difference in the mean data obtained from monaural or binaural animals as seen in

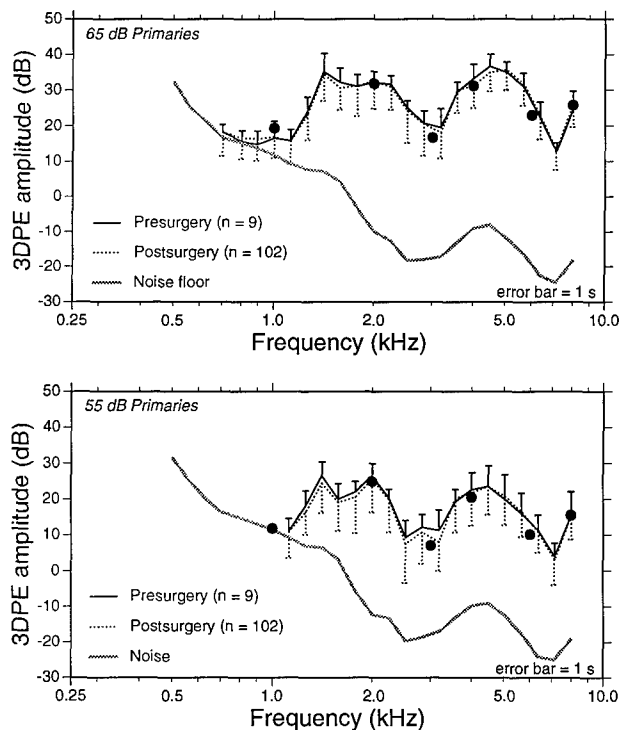


Figure 11. The normative DPEgram for the chinchilla before and after monauralizing surgery measured with 55 and 65 dB primaries. Solid data points at the indicated frequencies are 3DPE values taken from the normative I/O function at the appropriate primary levels. ( $s$  = one standard deviation)

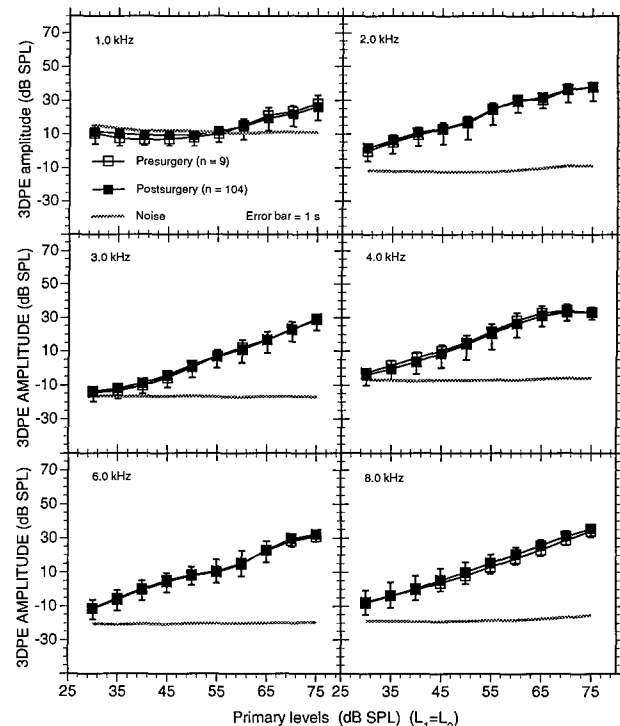


Figure 12. The normative I/O functions for the chinchilla at the indicated geometric mean of the primary frequencies before and after monauralizing surgery. ( $L_1$  = lower frequency primary level;  $L_2$  = upper frequency primary level;  $s$  = one standard deviation)



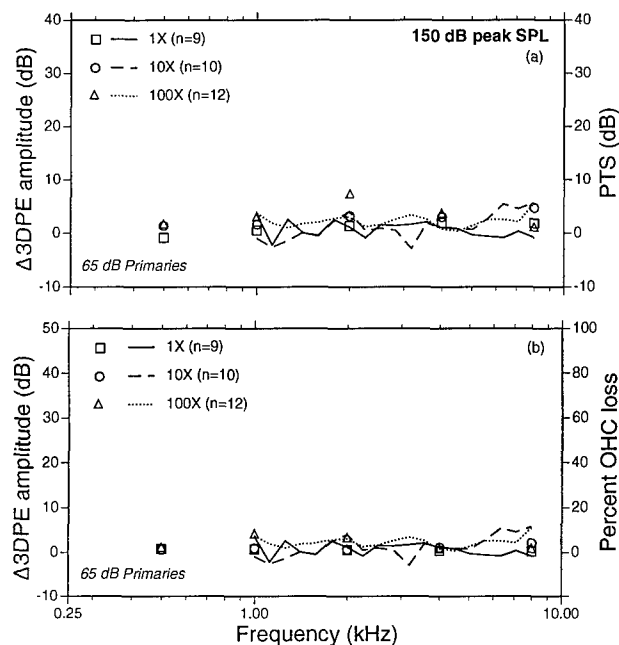


Figure 13. (a) The group mean difference in the pre- and postexposure DPEgram (lines) compared with the group mean PTS (symbols) and (b) compared to the group mean octave band outer hair cell (OHC) loss (symbols) for the three groups of animals exposed to the 150 dB peak SPL impulse.

Figures 11 and 12. The standard deviations of the test-re-test measurements which were based on 75 DPEgrams measured on each of nine binaural animals, over a period of 25-days was typically 5 to 6 dB. Following monauralizing surgery and recovery, the standard deviations, based on 15 data collection sessions over 5 days, in individual animals did not change. The 3DPE data have a frequency profile which is unique to each animal, while across animals the 3DPE at a given frequency can vary considerably. Thus, while the preexposure test-retest variability within animals was very small (5 to 6 dB over several days of testing) the standard deviations in the mean normative data are much larger; on the order of 10 dB. In all the data presentations that follow the standard deviation is shown by a vertical bar, which, when not present, indicates that it is smaller than the size of the symbol representing the data point.

**A) Postexposure Effects:** A summary of the group mean changes measured 30-days following the 150 and 155 dB peak SPL impulse noise exposures is presented in Figures 13 and 20 respectively. Each of these figures shows the group mean PTS and OHC percent loss measured over adjacent octave band lengths of the cochlea compared with the change in the 3DPE output using 65 dB primaries. This latter metric was obtained by computing the difference between the mean preexposure DPEgram and the mean postexposure DPEgram for each animal. Each difference DPEgram was then averaged across all animals in each exposure group to obtain the functions shown in Figures 13 and 20. There were no consistent differences in the effects of the exposures on the DPEgrams acquired with 55 or 65 dB SPL primaries. Inner hair cell loss data is not presented in these two figures since either there was none or it was much smaller than the OHC loss and localized to the region of the cochlea where the OHC loss was maximal. More detailed results of the histology and audiometry can be found in Ahroon et al. (1995).

**B) The 150 dB peak SPL results:** In general the three mean indices of trauma (PTS, 3DPEs and OHC loss) were not congruent across all exposure conditions. Exposure to the 1X or 10X, 150 dB peak SPL impulses (Figure 13) produced no PTS or sensory cell loss and there were no changes in the group mean data exceeding 10 dB in the DPEgrams or the I/O functions. However, recognizing

that this impulse has its greatest effect in the 1 to 4 kHz region, one animal (1876) in the 1X condition was particularly interesting. The cochleogram, audiogram and 3DPE data are shown in Figure 14. The animal had an essentially normal cochleogram and audiogram (i.e. within 10 dB) but in the 65 dB DPEgram the emissions were depressed in the 2 to 3 kHz region. The I/O functions were also depressed; most severely at 2 kHz (Figure 14, inset) and consistently 10 to 15 dB at 1, 3 and 4 kHz. Enhanced emissions, exceeding 10 dB of preexposure values, were measured in this subject below 1 kHz. Also note that the standard deviations of the 30-day post exposure I/O function shown, especially at the lower primary levels, are substantially increased over the preexposure values which are smaller than the size of the symbols used to present the data points. This was a frequent feature of the postexposure 3DPE data in animals that showed some effect of the noise. One other animal in this group showed similar I/O function changes between 1 and 4 kHz but only for primaries below about 50 dB. All the other animals in this group were unremarkable, displaying normal cochleograms, audiograms, and 3DPEs.

Increasing the energy of the 150 dB exposure by 10 dB through a ten-fold increase in number of impulses did not appreciably change the group mean results as seen in Figure 13. Group mean (N=10) audiograms, DPEgrams and sensory cell populations were normal. As in the 1X condition, some of the individual animals were instructive. Individual PTS audiograms were near normal (i.e., within 5 dB) in 7 of the 10 animals. Three animals showed as much as a 15 dB PTS at 11.2 or 16 kHz. OHC populations showed very small scattered losses and were about the same as those found in the 1X condition. Most of the changes were seen in the 3DPEs. Figure 15 shows the most affected animal (Chinchilla 1882). The 65 dB DPEgram shows a depression in the 2 kHz region consistent with the changes seen in two of the animals in the 1X group and additional depressions in the frequencies above 5 kHz. As will be seen in subsequent figures, severe cochlear lesions begin to develop at these frequencies as impulse energies are increased. The I/O functions in this animal were depressed at 2, 4 (Figure 15, inset), 6 and 8 kHz. Similar changes in the I/O functions (Figure 16) were seen in three other animals in this group.

At the 100X condition the mean data shown in Figure 13 indicates there is a slight (8 dB) group mean PTS at 2 kHz which probably represents a real effect since not only is this the frequency which was typically most effected by this impulse, but there is also a small group mean loss of OHCs in the 1 and 2 kHz region. The mean changes in both the 55 and 65 dB DPEgrams, the latter shown in Figure 13, were very small and not at all suggestive of any changes in the 2 kHz region. The individual animals, however, provide a very different perspective. Two of these animals were completely normal in all metrics. Six had normal audiograms through 11.2 kHz, OHC losses that were very small and scattered much like in the two groups previously discussed and various I/O functions between 2 and 8 kHz that were significantly depressed especially at the lower and presumably more diagnostic, primaries. Postexposure standard deviations were noticeably increased. A sampling of some of the most-affected I/O functions in four of these animals illustrating these changes is shown in Figure 17. The effects of the noise on two of the remaining four animals are shown in Figures 18 and 19. Animals 1887 (Figure 18) and 1862 (not shown) were similar; each had OHC lesions in the 1 to 2 kHz region, less than 20 dB PTS across the mid-frequencies, and depressed DPEgrams in the 1 to 2 kHz and in the 3 to 4 kHz regions. Animals 1867 (Figure 19) and 1869 (not shown) were similar; each had very low level OHC losses, a PTS that was close to or exceeded 30 dB at 2 kHz, and large depressions in the 3DPEs. In both of these animals, changes in

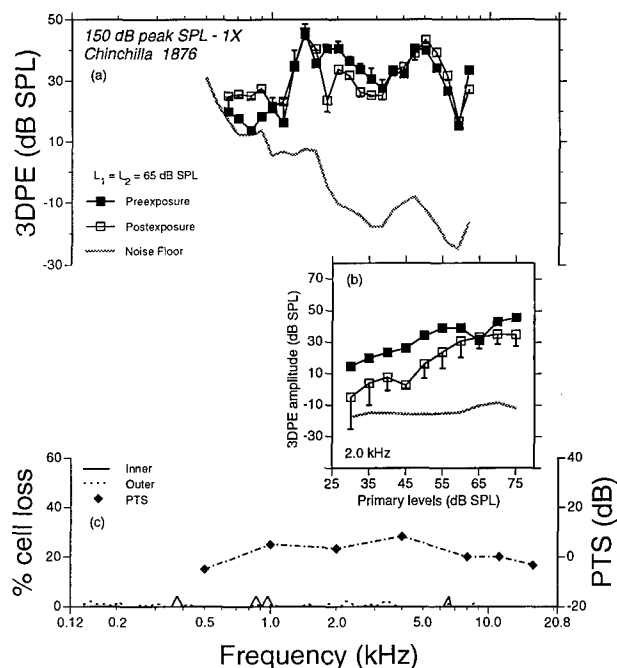


Figure 14. (a) The pre- and 30-day postexposure DPEgrams, (b) the pre- and 30-day postexposure 3DPE I/O function measured at 2.0 kHz, and (c) the cochleogram and PTS audiogram (symbols) for Chinchilla 1876 exposed to the 150 dB peak SPL, 1X, reverberant impulse.

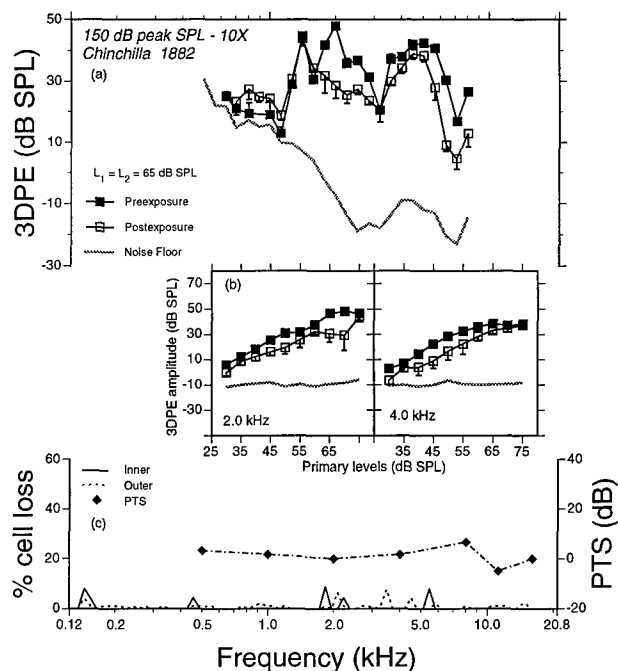


Figure 15. (a) The pre- and 30-day postexposure DPEgrams, (b) the pre- and 30-day postexposure 3DPE I/O functions measured at 2.0 and 4.0 kHz, and (c) the cochleogram and PTS audiogram (symbols) for Chinchilla 1882 exposed to the 150 dB peak SPL, 10X, reverberant impulses.

the 3DPEs generally paralleled the PTS audiograms. The extent of the PTS was surprising based on the limited OHC loss. In this particular example, the agreement between the PTS profile and the 3DPE data would indicate that sensory cell changes other than cell loss are responsible for the PTS and that the 3DPEs, in their agreement with the PTS, seem to be effective in picking up functional

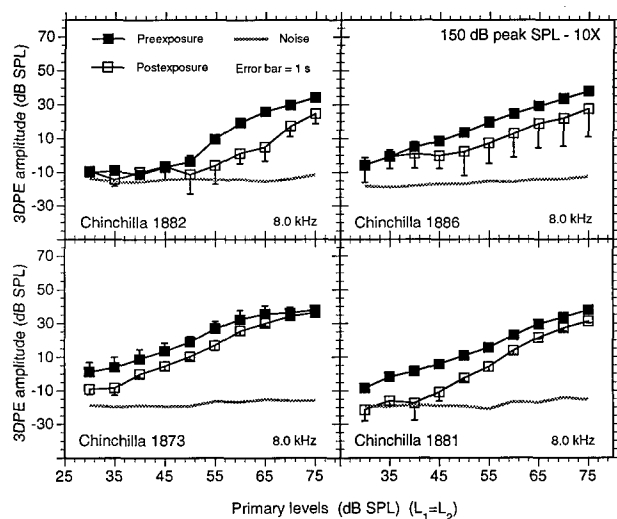


Figure 16. The pre- and 30-day postexposure 8.0 kHz I/O functions for four animals exposed to the 150 dB peak SPL, 10X, reverberant impulses.

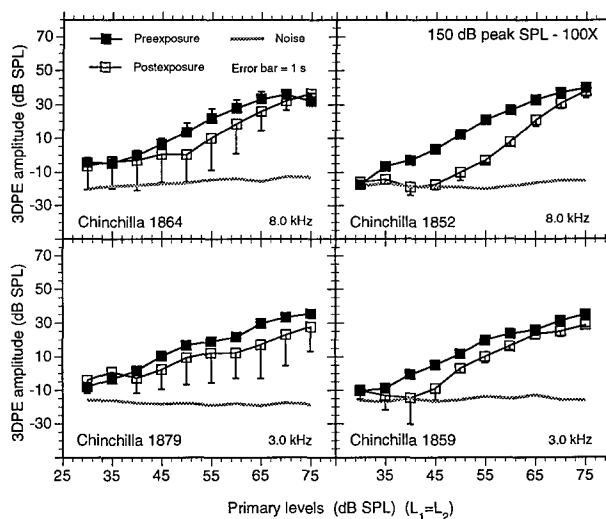


Figure 17. The pre- and 30-day postexposure 8.0 or 3.0 kHz I/O functions for four animals exposed to the 150 dB peak SPL, 100X, reverberant impulses.

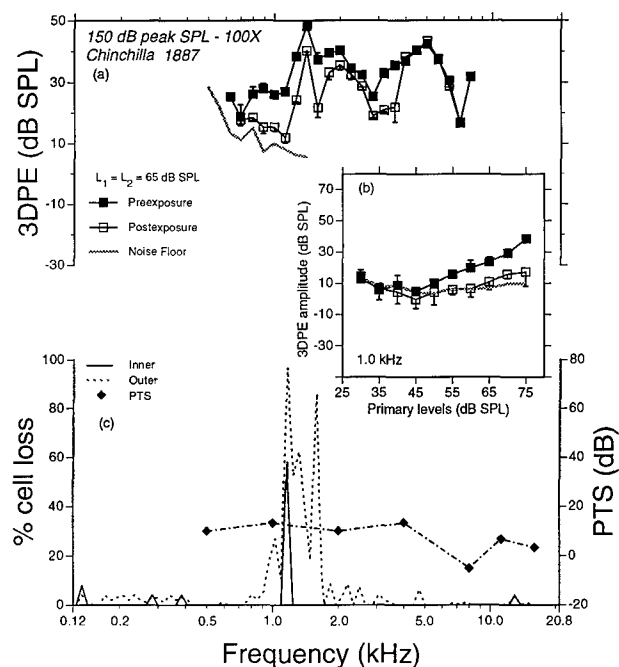


Figure 18. (a) The pre- and 30-day postexposure DPEgrams, (b) the pre- and 30-day postexposure 3DPE I/O function measured at 1.0 kHz, and (c) the cochleogram and PTS audiogram (symbols) for Chinchilla 1887 exposed to the 150 dB peak SPL, 100X, reverberant impulses.

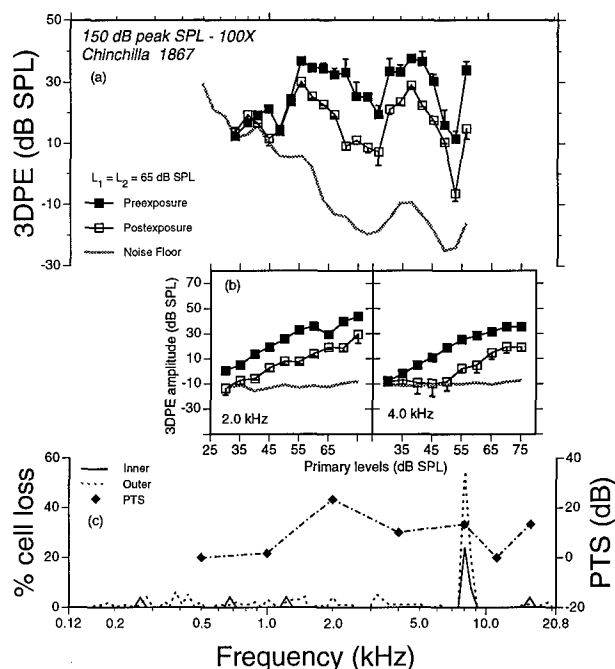


Figure 19. (a) The pre- and 30-day postexposure DPEgrams, (b) the pre- and 30-day postexposure 3DPE I/O functions measured at 2.0 and 4.0 kHz, and (c) the cochleogram and PTS audiogram (symbols) for Chinchilla 1867 exposed to the 150 dB peak SPL, 100X, reverberant impulses.

deficits that are not entirely the result of cell loss. This would also then suggest that the 3DPE depression in some of the 1X and 10X animals, where there was no PTS or cell loss, is indicative of altered sensory cell function.

C) The 155 dB peak SPL results: All of the results discussed above are more clearly or convincingly reinforced in the three groups exposed to the 155 dB impulse. The summary of the group mean data shown in Figure 20 indicates that for the 1X condition while there was, on average, no PTS, and no sensory cell loss, there were large and systematic changes in the 65 dB DPEgrams in the 1.5 to 3 kHz region. The 10X condition showed a group mean PTS that did not exceed about 3 dB at most frequencies, a group mean sensory cell population that was normal, but relatively large changes in the DPEgrams also in the 1.5 to 3 kHz range. The group mean difference DPEgrams for the 1X and the 10X conditions look surprisingly similar. The 100X condition produced very large changes in all indices of trauma which were consistent among the three metrics; PTS, 3DPEs and OHC loss.

Data from the two animals that made up the 155 dB, 1X group are shown in Figures 21 and 22. Both animals have normal thresholds, very minor sensory cell loss, and very large reductions in the 3DPE output. Animal 1790 showed 3DPE depression in the 1.5 to 3 kHz region while most of the frequencies above 1.5 kHz were depressed in Animal 1787. While the 10X condition produced, on average a small PTS, the cell loss and 3DPE changes were similar to the 1X condition. Animal 1786 shown in Figure 23 is typical of three animals in this group. Unlike Animal 1786, that had a small PTS of about 10 dB at the 8 and 11.2 kHz test frequencies, two of these animals had no PTS

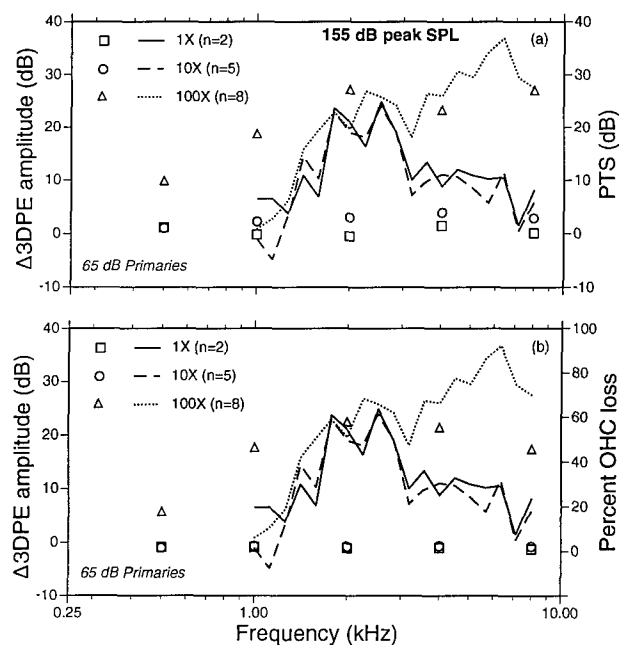


Figure 20. (a) The group mean difference in the pre- and postexposure DPEgram (lines) compared with the group mean PTS (symbols) and (b) compared to the group mean octave band outer hair cell (OHC) loss (symbols) for the three groups of animals exposed to the 155 dB peak SPL impulse.

at all. All three showed a consistent reduction in 3DPE output across the mid- and high frequencies. Animal 1792 (Figure 24) showed the largest PTS in the group but cell loss and 3DPEs were similar to those shown in Figure 23. One animal in this group showed no PTS or cell loss but did show a large depression of the 6 and 8 kHz I/O functions.

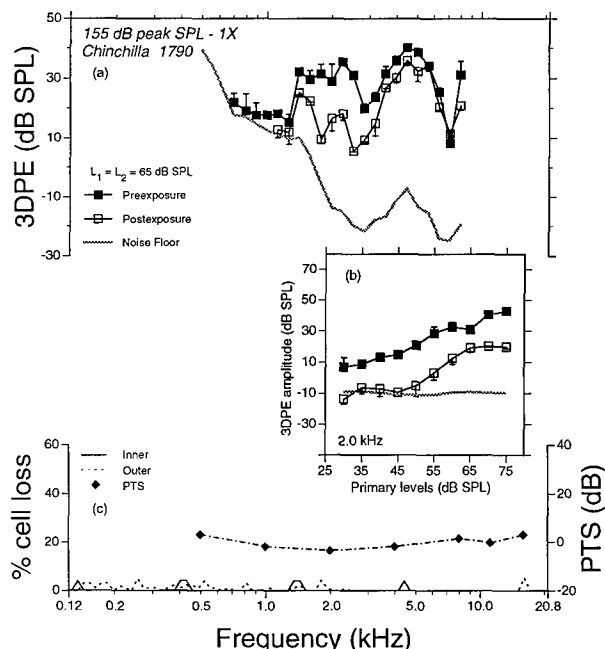


Figure 21. (a) The pre- and 30-day postexposure DPEgrams, (b) the pre- and 30-day postexposure 3DPE I/O function measured at 2.0 kHz, and (c) the cochleogram and PTS audiogram (symbols) for Chinchilla 1790 exposed to the 155 dB peak SPL, 1X, reverberant impulse.

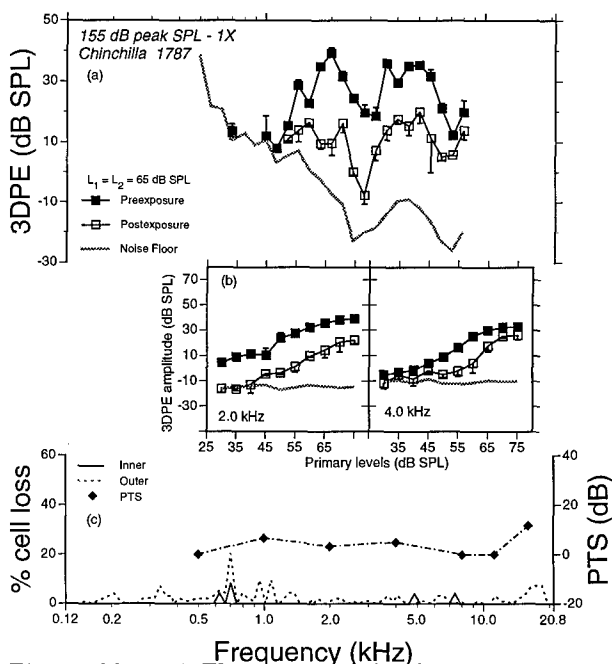


Figure 22. (a) The pre- and 30-day postexposure DPEgrams, (b) the pre- and 30-day postexposure 3DPE I/O functions measured at 2.0 and 4.0 kHz, and (c) the cochleogram and PTS audiogram (symbols) for Chinchilla 1787 exposed to the 155 dB peak SPL, 1X, reverberant impulse.

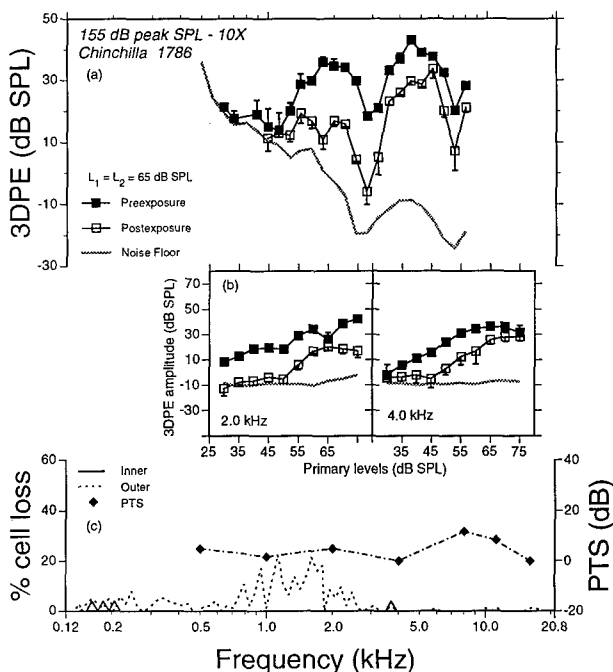


Figure 23. (a) The pre- and 30-day postexposure DPEgrams, (b) the pre- and 30-day postexposure 3DPE I/O functions measured at 2.0 and 4.0 kHz, and (c) the cochleogram and PTS audiogram (symbols) for Chinchilla 1786 exposed to the 155 dB peak SPL, 10X, reverberant impulses.

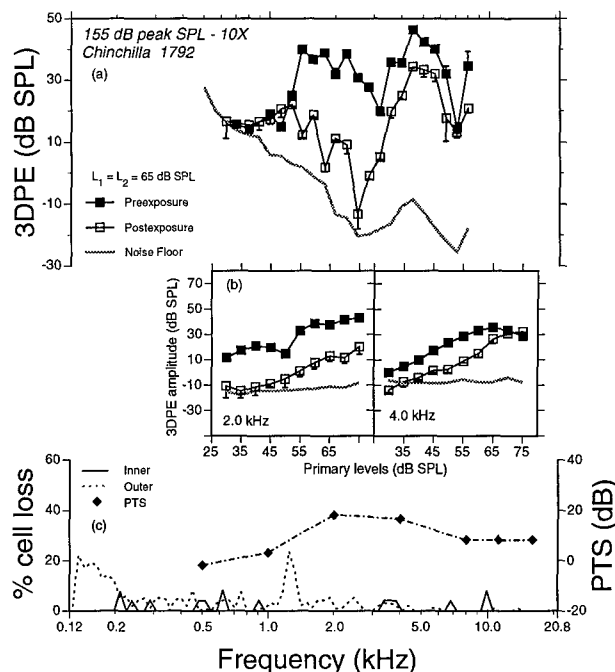


Figure 24. (a) The pre- and 30-day postexposure DPEgrams, (b) the pre- and 30-day postexposure 3DPE I/O functions measured at 2.0 and 4.0 kHz, and (c) the cochleogram and PTS audiogram (symbols) for Chinchilla 1792 exposed to the 155 dB peak SPL, 10X, reverberant impulses.

Six animals in the 155 dB, 100X group (N=8) showed severe sensory cell losses, large PTS at a number of frequencies, and severely reduced or absent emissions. Three of these animals having different levels of trauma are shown in Figures 25, 26, and 27. Animal 1798 (Figure 25) shows a bimodal sensory cell loss which is reflected in the 65 dB DPEgram which shows a loss around 1.5 kHz, normal 3DPEs between 2 and 3.5 kHz, and severe depression for frequencies above 4 kHz. The PTS audiogram shows severe losses only at the high (8 to 16 kHz) test frequencies. Animal 1793 (Figure 26) shows a severe OHC lesion between 1 and 8 kHz, maximum hearing loss at and above 8 kHz, and absent or severely depressed 3DPEs between 1 and 8 kHz. While the depression of 3DPEs in the 1-8 kHz region is not surprising, the relatively modest PTS of less than 10 dB below 8 kHz does not have a satisfactory explanation. Poor correlation among OHC loss and thresholds, however, are not unusual (Hamernik et al., 1989). This is further emphasized by comparison of OHC loss and PTS shown in Figure 27. Three animals in this group were similar to Subject 1796 whose data is shown in Figure 27. A nearly complete loss of OHCs from about 0.6 kHz to the basal end of the cochlea is accompanied by 40 to 50 dB PTS and severely depressed 3DPEs. One other animal was similar to Subject 1788 shown in Figure 28. Very little sensory cell loss was accompanied by a PTS of from 5 to 20 dB across the entire audiometric range and a fairly uniform reduction in 3DPEs across similar frequencies. In addition to the animals discussed above, one other animal was exposed to the same impulse but at 160 dB, 10X. The results of this exposure were virtually identical to the subject shown in Figure 27.

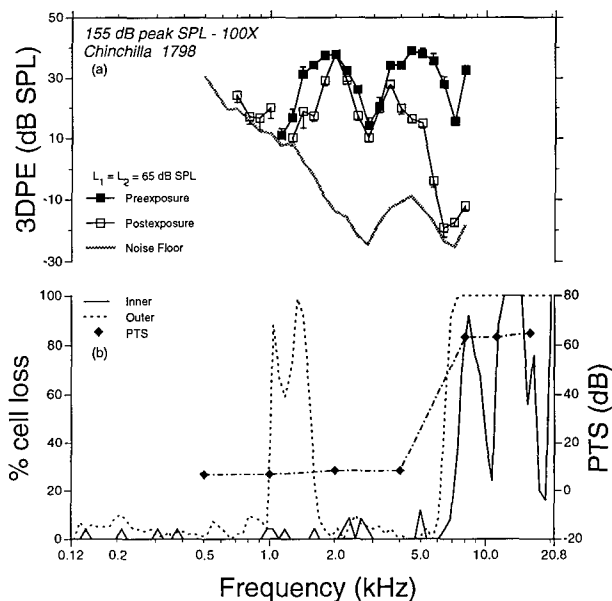


Figure 25. (a) The pre- and 30-day postexposure DPEgrams, and (b) the cochleogram and PTS audiogram (symbols) for Chinchilla 1798 exposed to the 155 dB peak SPL, 100X, reverberant impulses.

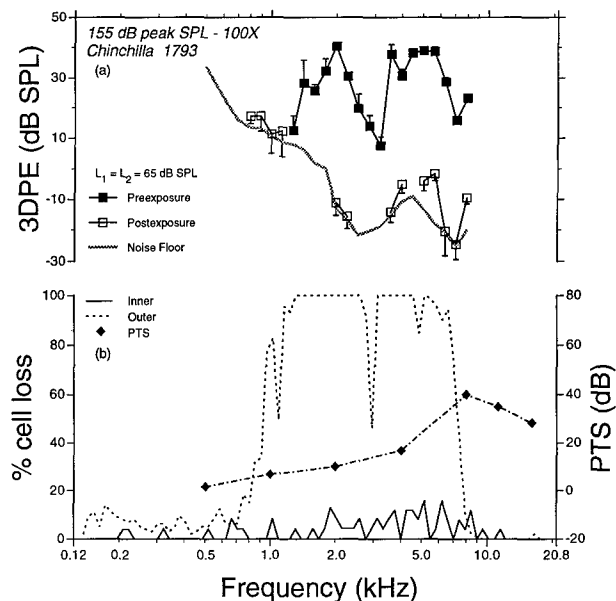


Figure 26. (a) The pre- and 30-day postexposure DPEgrams, and (b) the cochleogram and PTS audiogram (symbols) for Chinchilla 1793 exposed to the 155 dB peak SPL, 100X, reverberant impulses.

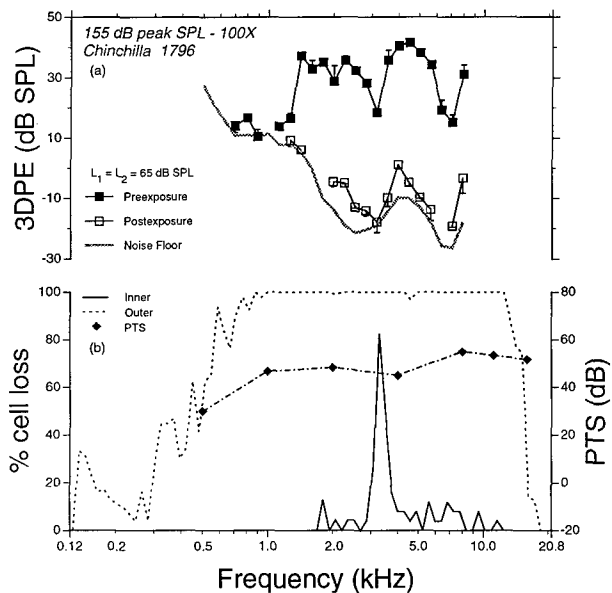


Figure 27. (a) The pre- and 30-day postexposure DPEgrams, and (b) the cochleogram and PTS audiogram (symbols) for Chinchilla 1796 exposed to the 155 dB peak SPL, 100X, reverberant impulses.

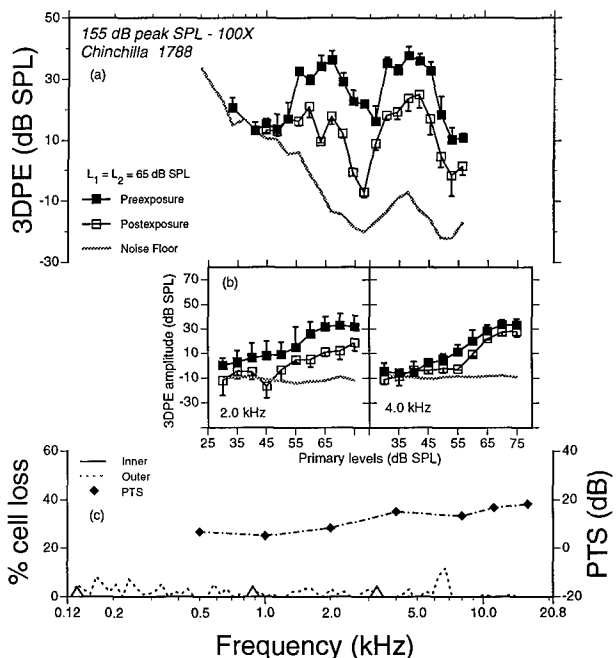


Figure 28. (a) The pre- and 30-day postexposure DPEgrams, (b) the pre- and 30-day postexposure 3DPE I/O functions measured at 2.0 and 4.0 kHz, and (c) the cochleogram and PTS audiogram (symbols) for Chinchilla 1788 exposed to the 155 dB peak SPL, 100X, reverberant impulses.

5) Discussion and Conclusions. In most of the early studies of noise-induced hearing loss, one of the underlying assumptions, in the effort to understand the hazards of the exposure, was that pure-tone thresholds were a reliable index of the effects of an exposure. Over the past 25 years a number of reports have uncovered difficulties with this assumption (see Hamernik et al., 1989, for a brief review). As a consequence, various psychoacoustic measures were proposed as diagnostic aids in the localization of noise-induced pathologies. Cochlear emissions testing is the most recent attempt to supplant or augment threshold testing for the specific diagnosis of outer hair cell pathologies. Unfortunately, clinical and animal-model studies are accumulating that show a variety of conflicting relations among thresholds, emissions, and cochlear pathologies (see Subramaniam et al., 1995, for an overview).

The results presented in this paper agree in principle with the conclusions of Brown et al. (1989) that changes in distortion product emissions can be associated with changes in the sensory cell morphology that may or may not lead to OHC loss. These 3DPE changes included increases in the variability of the postexposure, within-subject variability. Although we only obtained sensory cell population data, the inference that the 3DPE is a more sensitive and frequency-specific index of noise trauma than are threshold measures is supported by the data. It is clear, from the results of the 150 dB peak SPL exposures, that for this series of three lower-level exposure conditions the focus of sensory cell damage is in the region between 1 and 2 kHz (Figure 18), and that PTS begins to develop in this region. As exposure energies increase, cell loss spreads toward the high frequencies and PTS generally increases, but not in any consistent frequency-specific pattern. A number of the exposures that were used produced almost no cell loss in individual animals, and no PTS, but consistent changes in the 3DPEs measured 30 days after exposure at the 1-2 kHz frequencies which, as noise levels increase, are the first to be affected. This effect is also clearly seen in the group mean data (Figure 20) at the 1X and 10X, 155 dB conditions. In general, when there was OHC loss in restricted regions of the cochlea, PTS was not always measured but there were invariably changes in the 3DPEs at the appropriate frequencies. Furthermore, when there was a PTS and an OHC loss, the frequency profiles were not often coincident, while changes in the 3DPEs were remarkably so (Figure 25). For the most severe exposure conditions (e.g., Figure 27), PTS and 3DPEs were similarly shifted and related to the extent of OHC loss.

Finally it should be noted that only with a DPEgram resolution of several points/octave was it possible in some animals to detect changes regardless of the primary levels. I/O functions separated by an octave could be normal while the 3DPEs for some frequencies within the band shifted more than 10 dB. Thus, some of the discrepancies between 3DPE data and cell pathology discussed in the literature may be the result of insufficient frequency resolution in the 3DPE collection paradigm.



**C. Comparison of Audiograms Determined Using Pure Tone and One-third Octave Bands of Noise as Stimuli for the Chinchilla. (Published as USAARL Rpt. No. 94-50).**

1. Introduction: The chinchilla audiometric procedure currently in use for noise hazard studies at the U.S. Army Aeromedical Research Laboratory (USAARL) at Fort Rucker, Alabama, uses pure tone stimuli in a sound field test environment (Patterson et al., 1986). The animals are trained in a shuttlebox to respond to sounds by moving from where they are to the other end of the shuttlebox. They are free to move about the test cage throughout the test. The tonal test stimuli are presented by a speaker located in one corner of an audiometric room with sound absorbing walls, thus creating a progressive and directional sound field. The animals are typically monaural. This leads to a possibility that they may orient their "hearing" ear differently with respect to the sound source from time to time. Since the orientation of the head relative to a sound source affects the level reaching the ear, the threshold will vary as the orientation is changed. This adds to the measurement error and is a source of uncontrolled variability in the threshold shifts measured in noise exposure studies.

An obvious solution to this problem is to always orient the subject's ear toward the sound source. One way to assure that the subject is always orienting the ear of interest toward the sound source is to make the source surround the subject. This can be done by using a quasireverberant test room to produce a nondirectional sound field. The ANSI standard method for real ear attenuation (ANSI, 1984) is based on this type of sound field. It uses one-third octave bands of noise originating from three speakers in a hard walled room as audiometric test stimuli. This study was undertaken to determine whether an audiometric test procedure for the chinchilla based on this quasireverberant sound field will lead to more reliable threshold estimates.

2. Methods: The subjects for this study were five male chinchillas from the USAARL chinchilla colony. They were monauralized by surgical destruction of the left cochlea. The surgery was done with the animal anesthetized by isoflurane gas inhalation. Aseptic procedures were followed during surgery. At least one week recovery was allowed after surgery before audiometric training or testing was conducted.

The audiometric testing employed a shock-avoidance procedure in a two-compartment shuttlebox (Patterson et al., 1986). The one-third octave band stimuli had center frequencies at 0.125, .25, 0.5, 1.0, 1.6, 2.0, 3.15, 4.0, 6.3, and 8.0 kHz. Each subject was trained in the audiometric procedure using one-third octave bands of noise until their thresholds reached asymptote. Then, 20 additional audiograms were obtained using the noise stimuli. This was followed by five audiograms using pure-tone stimuli for transitional training. Finally, 15 to 20 pure-tone audiograms were obtained. The pure-tone stimuli had frequencies of 0.125, .25, 0.5, 1.0, 1.6, 2.0, 3.15, 4.0, 6.3, and 8.0 kHz.

The 15 to 20 audiograms from each type of test stimulus were used to calculate a test-retest variance estimate for each subject (except for one subject who died before completing the pure-tone test) at each test frequency. Under the null hypothesis that there is no difference in the test-retest variability of these two procedures, the ratio of these variance estimates is distributed as F (Brownlee, 1960). This test was used to determine the significance of differences in test-retest reliability of the two procedures.

3. Results and discussion: Table 2 contains the average audiometric thresholds determined using the one-third octave band stimuli and the standard deviation of these thresholds for each of the five subjects. These results are based on 20 repeated determinations of threshold for each subject. Table 3 contains the corresponding results from the pure-tone audiometry for the same subjects.

**Table 2**

Average values and standard deviations of the thresholds determined using one-third octave bands of noise.

Subject	Frequency in kHz									
	0.125	0.250	0.50	1.0	1.6	2.0	3.1	4.0	6.3	8.0
X63	21.8	13.0	2.5	2.3	1.3	2.0	-3.5	-1.8	0.8	-1.5
s	8.8	4.2	4.2	5.8	5.7	5.7	3.7	6.8	7.8	3.7
X64	20.5	10.0	-1.3	-21.8	1.8	-1.3	0.3	-0.3	2.8	-0.3
s	4.3	6.0	5.0	5.6	5.3	3.1	4.9	3.3	6.2	3.3
X66	21.8	13.8	0.5	-1.0	-3.3	-1.3	1.0	-5.3	-1.3	-0.5
s	4.5	4.5	7.0	5.9	5.3	4.1	6.9	4.0	5.4	4.0
X68	19.3	11.0	1.5	-2.8	0.0	-2.5	0.3	2.8	1.8	2.0
s	4.5	5.7	6.0	5.4	6.6	5.2	7.2	5.6	6.8	5.7
X55	19.5	9.3	-4.3	-3.9	-0.8	0.9	1.8	1.3	3.5	5.8
s	5.3	4.5	5.5	5.2	5.8	5.8	7.6	4.4	5.4	3.3
Mean	20.6	11.4	-0.2	-5.4	-0.2	-0.4	0.0	-0.7	1.5	1.1
s	1.2	1.9	2.7	9.4	2.0	1.8	2.0	3.1	1.9	2.9

**Table 3**

Average values and standard deviations of the thresholds determined using pure tone stimuli.

Subject	Frequency in kHz										n
	0.125	0.250	0.50	1.0	1.6	2.0	3.1	4.0	6.3	8.0	
X63	21.8	5.3	-3.3	0.3	-4.3	-2.5	3.0	-3.5	-1.0	-0.5	20
s	7.5	6.4	6.6	8.6	10.3	8.2	9.7	6.2	8.2	7.6	
X64	17.5	2.5	-5.8	-8.8	-5.0	-2.8	1.3	-2.3	1.5	-2.3	20
s	4.5	8.1	6.8	4.7	5.8	6.8	4.7	6.4	9.0	6.6	
X66	22.8	6.5	-7.5	-3.5	0.2	-6.5	-3.2	-2.2	1.5	1.8	15
s	5.3	3.7	5.2	3.7	5.4	4.9	6.5	5.3	8.0	7.5	
X68	19.8	9.5	-3.2	-6.2	1.2	-3.8	1.5	0.5	1.5	7.2	15
s	10.1	3.1	7.3	5.0	5.3	5.6	5.8	8.1	6.9	5.9	
X55	17.5	-1.6	-1.1	-5.2	-10.2	-5.7	2.0	4.8	-2.0	4.3	10
s	7.7	8.7	7.1	5.8	2.5	6.5	7.5	7.2	6.2	6.1	
Mean	19.9	4.4	-4.2	-4.7	-3.6	-4.3	0.9	-0.5	0.3	2.1	
s	2.4	4.2	2.5	3.4	4.6	1.8	2.4	3.3	1.7	3.8	

The number of pure tone thresholds for each subject is indicated since not all subjects were tested 20 times. Table 4 contains F-ratios resulting from dividing the variance of the pure-tone thresholds by the variance of the one-third octave band thresholds. These F-ratios have 19 degrees of freedom (DF) in the denominator and the numerator DF are as indicated in the table. Large values of F indicated that one-third octave band thresholds are more reliable. The F-ratio is significant at the 0.05 level in 13 cases out of 50, or 26 percent of the cases. Inverting the F-ratios will test for the cases where the pure-tone thresholds are more reliable. The F-ratios are significant at the 0.05 level in three cases, or only 6 percent of the cases. This overall pattern of variance ratios indicates that the audiometric procedure using one-third octave bands represents some improvement in the test-retest reliability of the thresholds. This effect is most pronounced in Subject X63 that showed increased reliability at most frequencies. In contrast, Subject X68 showed no improvement in reliability with the one-third octave band stimuli.

**Table 4**

F-ratios formed by dividing the test-retest variance of thresholds determined using pure-tone stimuli by thresholds determined using one-third octave bands of noise. (ndf = numerator degrees of freedom)

Subject	Frequency in kHz										ndf
	0.125	0.250	0.50	1.0	1.6	2.0	3.1	4.0	6.3	8.0	
X63	0.71	2.39*	2.47*	2.19*	3.28*	2.09	6.77*	.085	1.12	4.18*	19
X64	1.08	1.79	1.85	0.71	1.20	4.77*	0.94	3.68*	2.11	3.91*	19
X66	1.36	0.71	0.55	0.40†	1.05	1.40	0.90	1.74	2.16	3.51*	14
X68	4.97	0.28†	1.45	0.87	0.65	1.15	0.66	2.12	1.04	1.08	14
X55	2.07	3.70*	1.64	1.23	0.19†	1.27	0.97	2.62*	1.33	3.52*	10

\* 1/3 octave band thresholds significantly more reliable at the .05 level  
† pure tone thresholds significantly more reliable at the .05 level

Figure 29 compares the average audiograms determined by the two procedures. Since the one-third octave band stimuli are relatively narrow band, we would expect that band levels at threshold to agree with the pure tone levels at threshold. Generally, the agreement is fair. There are a few frequencies at which the thresholds seem to differ (e.g., 0.25 through 2.0 kHz); however, these differences are not large (4 to 7 dB). An analysis of variance (Winer, 1962) was used to test for equivalence of the average audiograms determined by the two methods. The main effect for stimulus type was significant at the .05 level, indicating there was a difference in the SPL at threshold between the two types of audiograms. In addition, the frequency by stimulus type interaction was significant indicating that differences were not consistent at all frequencies.

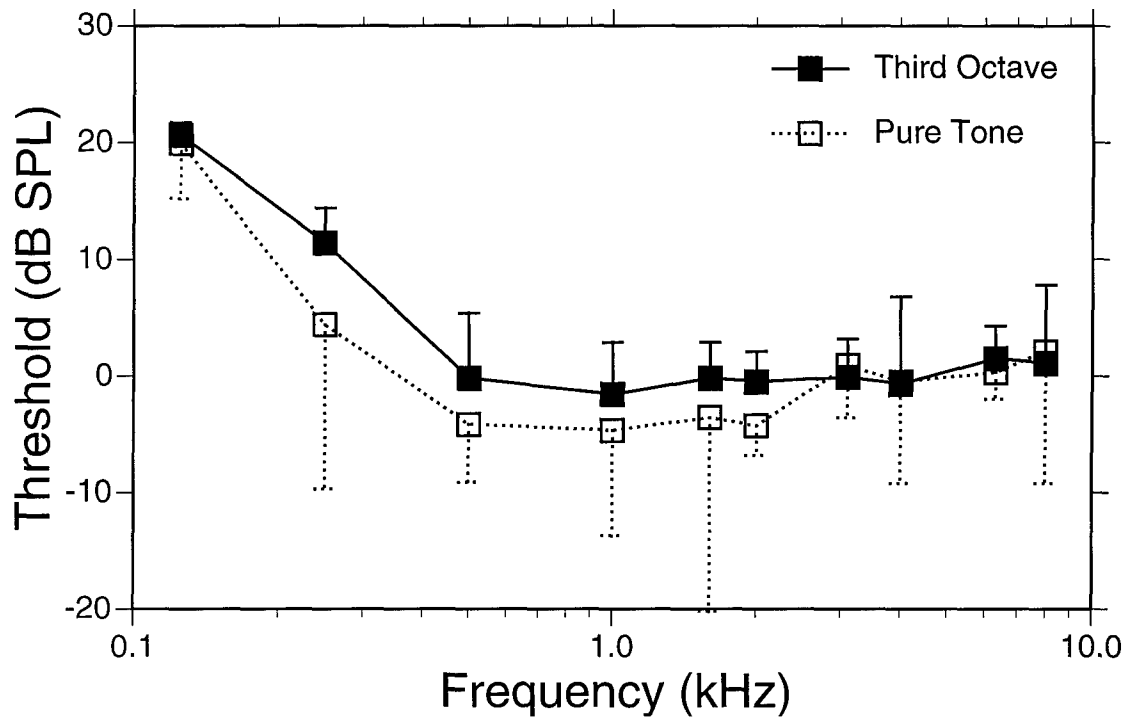


Figure 29. Average threshold in sound pressure level determined using pure tone and 1/3 octave bands of noise stimuli.

4. Conclusions: The audiograms determined using one-third octave bands of noise in a quasireverberant sound field are at least as reliable as the conventional pure-tone audiograms for the chinchilla. The sound pressure levels at threshold are different at least at some frequencies.

#### D. The cubic distortion product otoacoustic emissions (3DPE): A normative data base.

In addition to the above study of the normative and the blast wave-induced changes in the cubic distortion product otoacoustic emissions using the Virtual model 330 instrument, the development of a normative data base using the Etymotic ER-10C instrument with CUB<sup>®</sup>DIS V2.40 software was initiated at both the SUNY Auditory Research Laboratories and the USAARL facilities. The objectives were: 1) to compare normative data samples across the two laboratories using similar instruments, and 2) to look at the effect of systematic variations in the level of the primary tones. This latter effort was undertaken in order to determine the levels of the two primary tones that would yield the most robust emissions (Whitehead et al., 1995a,b), and thus a test paradigm that would be most sensitive to blast wave-induced cochlear pathologies. This work was not completed. However, since the results of this data collection effort may be instructive, they are included with this report without substantive comment. When instructions were received to terminate all animal experimentation associated with this contract no further experimental animal work was performed.

Figure 30(a) shows the mean cubic distortion product emissions (3DPE) collected on 77 non-noise-exposed animals in the SUNY ARL facilities. These animals were subjected to only the surgical treatment required to ablate the left cochlea (monauralization) and to implant the AEP electrode as described previously. In this data collection paradigm the primary levels were kept equal ( $L_1=L_2$ ) and  $f_2/f_1=1.22$ . The primary levels were varied from 20 dB to 70 dB SPL in 10 dB steps. At the 20 dB primary level the 3DPEs are in the noise floor. For the higher levels there is a systematic and approximately linear increase in emissions with primary level. These data generally agree with published data on the chinchilla. Figure 30(b) shows the mean 3DPEs collected on a normal (except for surgery as indicated above) group of 20 chinchillas in which the primaries were offset by 10 dB, i.e.  $L_1=L_2+10$  dB. A 10 dB offset was chosen based on the results of Whitehead et al., (1995a) showing optimal emissions for this condition.

A parallel data set obtained at the USAARL facility using a similar emissions collection protocol is shown in Figures 31(a) and (b). These data were collected on a sample of 18 chinchillas. In Figure 31(a) the primary levels were equal and varied from 20 dB to 55 dB SPL in 5 dB steps,

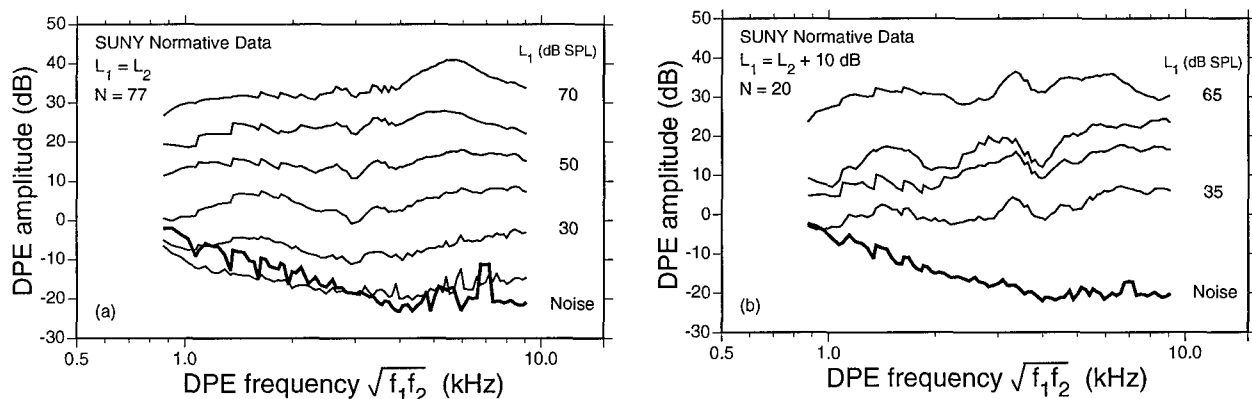


Figure 30. The mean cubic distortion product otoacoustic emissions measured at the SUNY ARL facility using (a) equal primary levels ( $L$ ) and (b) primary levels offset by 10 dB.

while in Figure 30(b) the primaries are offset by 10 dB. A summary of all the individual animal emissions data collected at the USAARL is shown in Appendices A and B.

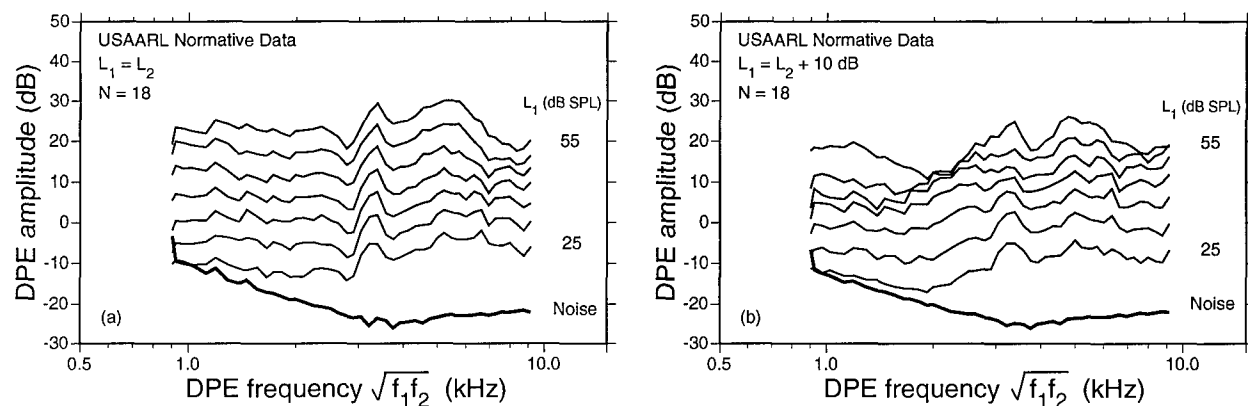


Figure 31. The mean cubic distortion product otoacoustic emissions measured at the USAARL facility using (a) equal primary levels ( $L$ ) and (b) primary levels offset by 10 dB.

## E. The prediction of PTS from a P-weighted energy model of impulse noise induced hearing loss

1. Background: The objective of this study, which was not completed, was to test the predictive value of the isohazard function developed for impulse noise by Patterson et al., (1993) when applied to actual blast waves. This function, which is an empirically derived relation, extracted from a data base obtained using synthetic impulses, defines a P-weighted energy metric. Since the anatomical correlates of exposure to the synthetic impulses which were used to derive the function were shown to be similar to the effects of actual blast waves (see Section A) the isohazard function should then theoretically be able to predict the auditory effects of a known blast wave exposure.

To test the feasibility of such an approach to predicting hazard, a series of shock tube exposures were designed having a total P-weighted sound exposure level that would produce a desired level of PTS based on the isohazard function of Patterson et al., (1993). The first and only exposure in this planned series consisted of an exposure to twelve, 157 dB peak SPL blast waves presented 3/min. This exposure was estimated on the basis of Patterson et al., (1993) to produce a mean PTS across the 1,2, and 4 kHz audiometric test frequencies of about 20 dB.

2. Methods: The experimental protocols were explained in some detail at the beginning of this report. Briefly, three groups of experimental animals were used. Group 1 consisted of 10 chin-chillas each individually exposed to 12 shock tube generated blast waves presented 3/min at 157 dB peak SPL. A spectrum and pressure-time history of the blast wave is shown in Figure 32. Each animal was monauralized and preexposure behavioral audiograms and emission functions collected on each animal. The animal was exposed and following exposure pure tone thresholds were remeasured immediately after exposure and ten more times over the thirty-day recovery period. Cochlear emissions were also collected on Day 1, 7,14, 21 and 30 following exposure. From this

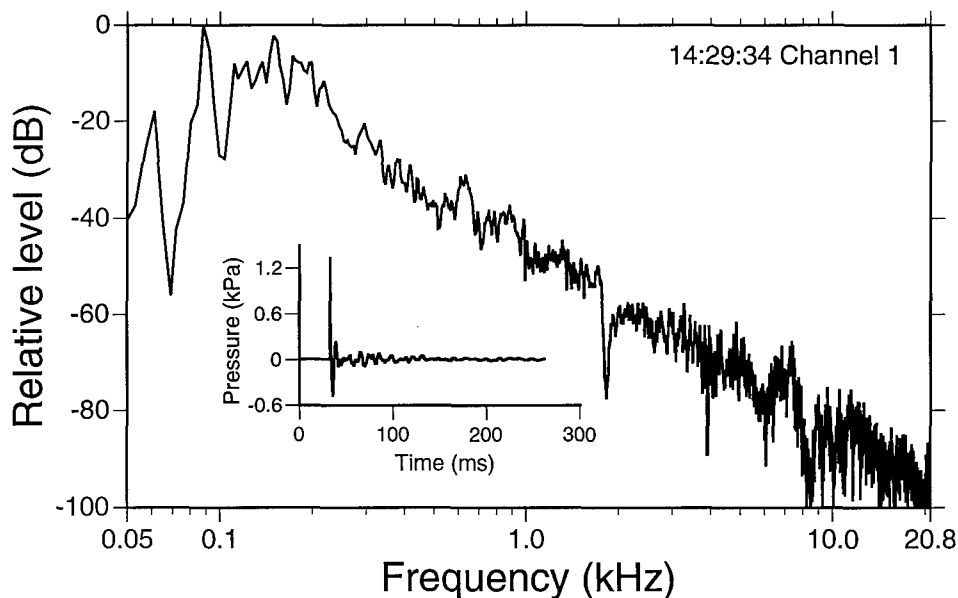


Figure 32. The pressure-time waveform and spectrum of the 157 dB peak SPL broad-band impulse.

protocol recovery functions and permanent changes in these metrics could be obtained. Following the final test procedure, the animals were euthanized and the cochleas prepared for the surface preparation histology. Cochleograms were prepared for each cochlea. A complete data summary for this group can be found in Appendix A.

Group 2 consisted of nine chinchillas that comprised the control group. These animals were subjected to a similar experimental protocol as the group 1 animals except for the actual blast wave exposure. A complete data summary for this group can be found in Appendix B.

Group 3 consisted of 19 chinchillas for which only quantitative histological data (cochleograms) were obtained. A complete data summary for this group can be found in Appendix C.

3. Results: The group mean preexposure audiogram and PTS are shown in Figure 33. Mean PTS across all test frequencies was less than 10 dB. Figure 34 shows the group mean sensory cell loss. Mean outer hair cell losses were approximately 20 % across the 1 to 10 kHz region of the cochlea. Initial mean postexposure threshold shifts, shown in recovery curves presented in Figure 35, varied from about 15 dB at the lower test frequencies to about 35 dB at the mid frequencies. Based on previous work, the mid-regions of the cochlea are typically first affected by these blast waves. The recovery functions were typical with only the 6.3 kHz test frequency showing some TS growth. The group mean 3DPEs are shown in Figures 36 and 37. Emissions obtained with both equal primaries and with primaries offset by 10 dB showed reductions of less than 10 dB which were consistent across frequency.

A review of the cochleograms shown in Appendices A and C will serve to emphasize the large individual variability that was found following this blast wave exposure. Compare, for example, subject X78 and X85 shown in Appendix C. The former shows virtually no sensory cell loss while the latter shows a complete loss of outer hair cells over almost 50% of the organ of Corti. This is a problem, that despite rigorous control of stimulus presentation variables, continues to plague impulse noise studies and has been the subject of numerous discussions in the literature. To date there is no adequate explanation for such an extreme individual susceptibility. Median data such as

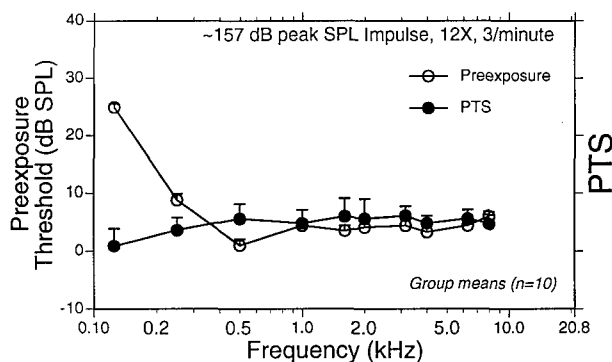


Figure 33. The mean preexposure audiogram for Group 1 subjects (O) along with the PTS measured 30 days following exposure to the 157 dB peak SPL blast waves.

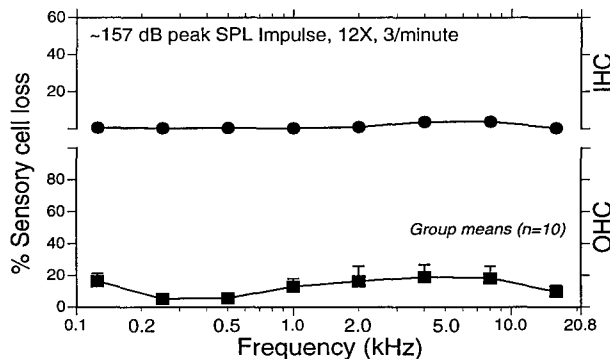


Figure 34. The Group 1 mean inner (IHC) and outer (OHC) hair cell loss following recovery from the 157 dB peak SPL blast wave exposure.



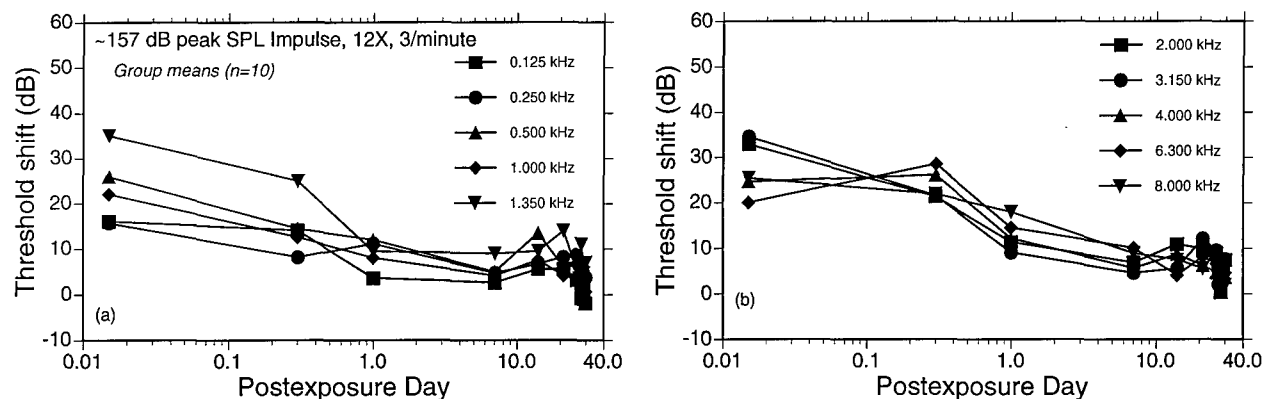


Figure 35. The mean threshold shift recovery curves for Group 1 subjects measured from immediately following exposure and at various points to 30 days postexposure.

that presented in Figures 33 through 37 thus often fail to convey the effects of exposures that produce such a bimodal effect. Another confounding result often reported in studies of noise trauma is the lack of agreement between pure tone thresholds and the status of the sensory cell population in the cochlea. Surveying the individual animals presented in Appendix A shows examples of subjects (e.g. Z73) that have large PTS (10 to 20 dB) but little or no sensory cell loss, while at the opposite extreme are the subjects (e.g. A38) that have large losses of sensory cells with little (< 10 dB) or no PTS. Also, for most subjects the frequency specific profile of PTS and the pure tone audiogram are not congruent further highlighting the lack of agreement among the two metrics.

Figures 38(a), (b) and (c) compare the audiometric, histological and emissions data from three individual subjects. In each case the depressions in the 3DPEs appear to correlate with the location and extent of the outer hair cell loss. Based on these preliminary results and the data presented in Section B of this report, the 3DPEs appear to be a more sensitive and frequency specific index of the blast wave-induced pathology.

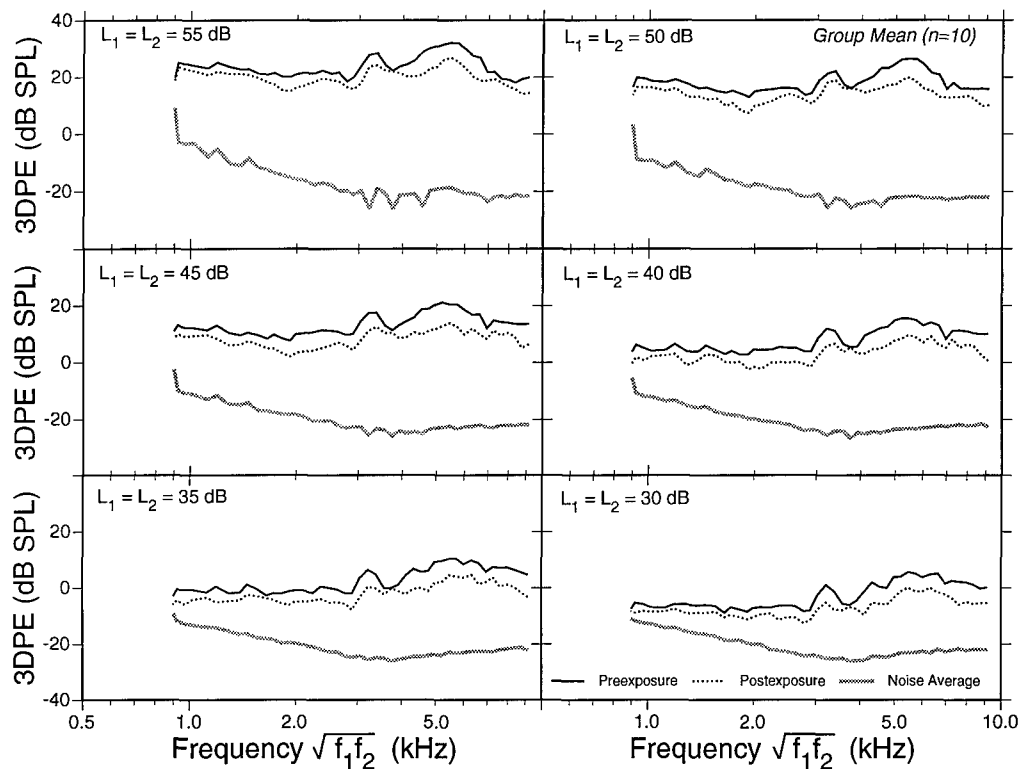


Figure 36. The mean pre- and postexposure cubic distortion product otoacoustic emissions measured on the Group 1 subjects using equal primary levels.

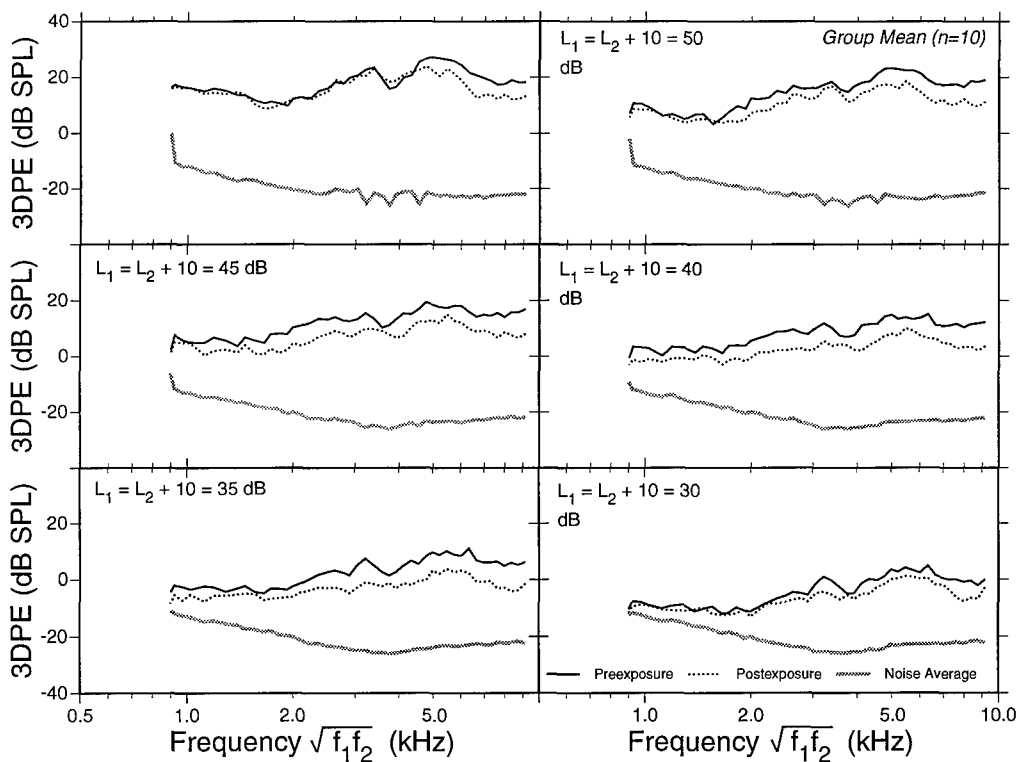


Figure 37. The mean pre- and postexposure cubic distortion product otoacoustic emissions measured on the Group 1 subjects using primary levels offset by 10 dB.

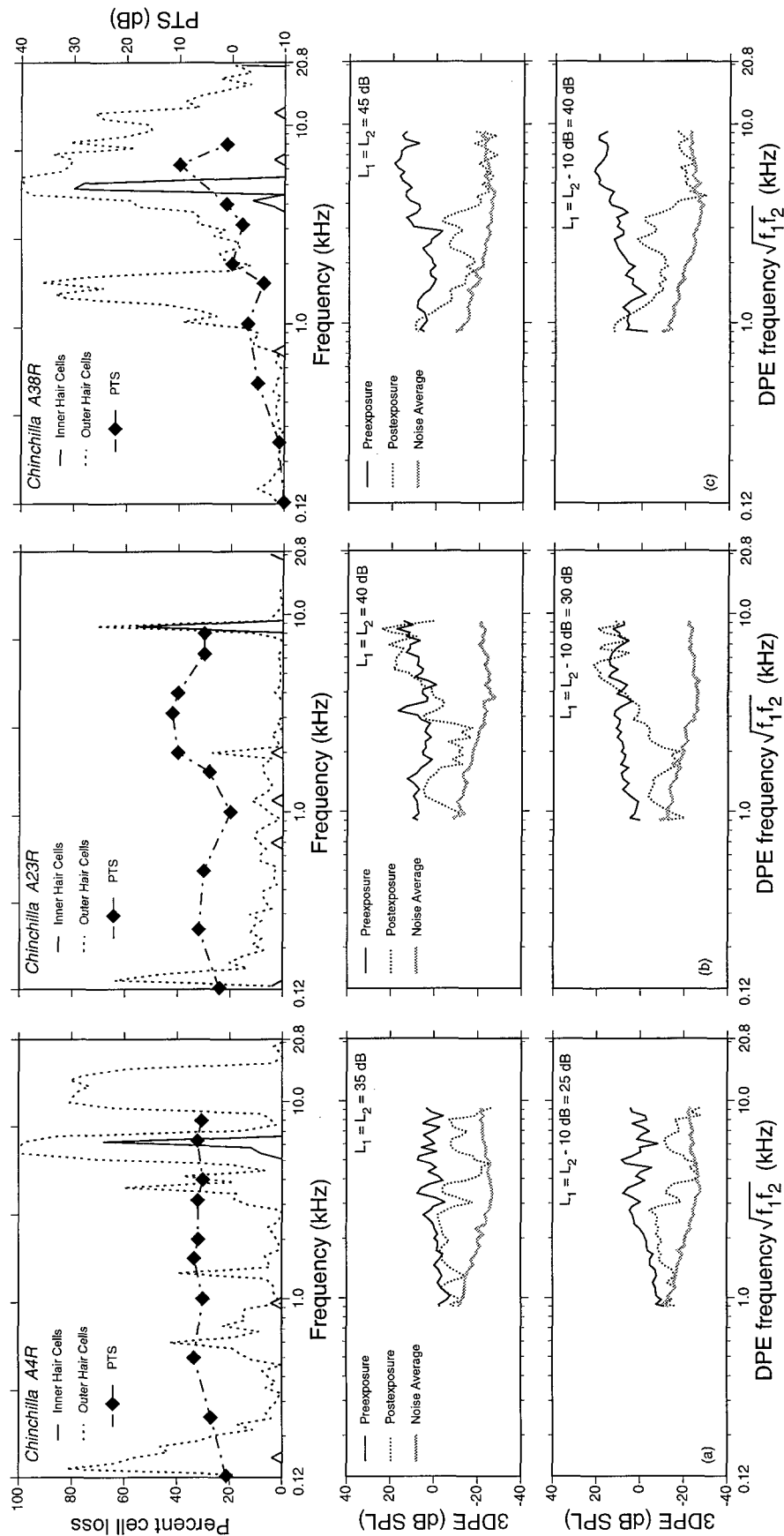


Figure 38. A selection of individual animal cochleograms and PTS audiograms (top panels); and cubic distortion product otoacoustic emissions (lower two panels).

## V. Acknowledgments

We gratefully acknowledge the following persons for their assistance in the conduct of this research: Laurie G. Aldrich, Derrick Arquiett, Sukhdev R. Bawa, Pat Bridges, Corinne Carey, Christopher Case, Stephen Fronczak, Sheldon E. Hager, DVM, C.E. Hargett, Brenda M. Jock, Ann R. Johnson, Sheau-Fang Lei, Renee Lincoln, Laural Mitchell, James B. Nichols, DVM, Kristen L. Petriello, Kelsey Seeley, and George A. Turrentine.

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## **VII. Bibliography of All Publications and Meeting Abstracts**

### **A) Publications:**

- Hamernik, R.P., Patterson, J.H., Jr., Case, C., and Hargett, C.E. (1996). Exposure to high level impulse noise: anatomical correlates. Audiology and Neuro-Otology. (submitted)
- Patterson, J.H., Jr., and Hamernik, R.P. (1996) Blast overpressure induced structural and functional changes in the auditory system. Toxicology. (in press).
- Patterson, J.H., Jr., and Hamernik, R.P. (1992) An experimental basis for the estimation of auditory system hazard. in Noise-induced Hearing Loss, eds., A.L. Dancer et al., Mosby Year Book, St. Louis, MO. pp 336-348.
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- Hamernik, R.P., Ahroon, W.A. and Lei, S-F. (1996) The cubic distortion product otoacoustic emissions from the normal and noise-damaged chinchilla cochlea. J. Acoust. Soc. Am. 100; 1003-1012.

### **B) Presentations:**

- Patterson, J.H., Jr. (1994) Blast overpressure induced auditory alterations. Workshop on the molecular mechanisms of blast overpressure induced injury, 26 Oct 94, Washington, D.C.
- Hamernik, R.P., Ahroon, W.A. and Lei, S-F. (1996) The cubic distortion product otoacoustic emissions from the normal and noise-damaged chinchilla cochlea. Assoc. for Res in Otolaryngol. 4-8 Feb 1996, St. Petersburg Beach, FL., Abs. 106, p.27.

# **VIII. List of Personnel Receiving Pay From the Negotiated Effort**

<u>Name</u>	<u>Title</u>	<u>Role in Project</u>
Roger P. Hamernik	Professor	Principal Investigator
William A. Ahroon	Sr. Research Scientist	Investigator
Laurie G. Aldrich	Research Lab Worker	AEP Technician
Sukhdev R. Bawa	Sr. Research Support Specialist	Histology Technician
Pat Bridges	Clerk	Secretary
Corinne Carey	Research Technician	Histology Technician
Christopher Case	Sr. Research Support Specialist	Histology Technician
Stephen Fronczak	Sr. Research Support Specialist	Histology Technician
C.E. Hargett	Sr. Research Scientist	USAARL Facility (on site rep.)
Brenda M. Jock	Sr. Research Aid	AEP Technician
Sheau-Fang Lei	Research Scientist	Acoustics Laboratory
Renee Lincoln	Clerk	Secretary
Laural Mitchell	Sr. Research Aid	Histology Technician
Kristen L. Petriello	Research Aid	Histology Technician
Kelsey Seeley	Research Aid	AEP Technician
George A. Turrentine	Project Support Specialist	Histology Technician



## APPENDIX A

Summary Data for the Group Exposed to:

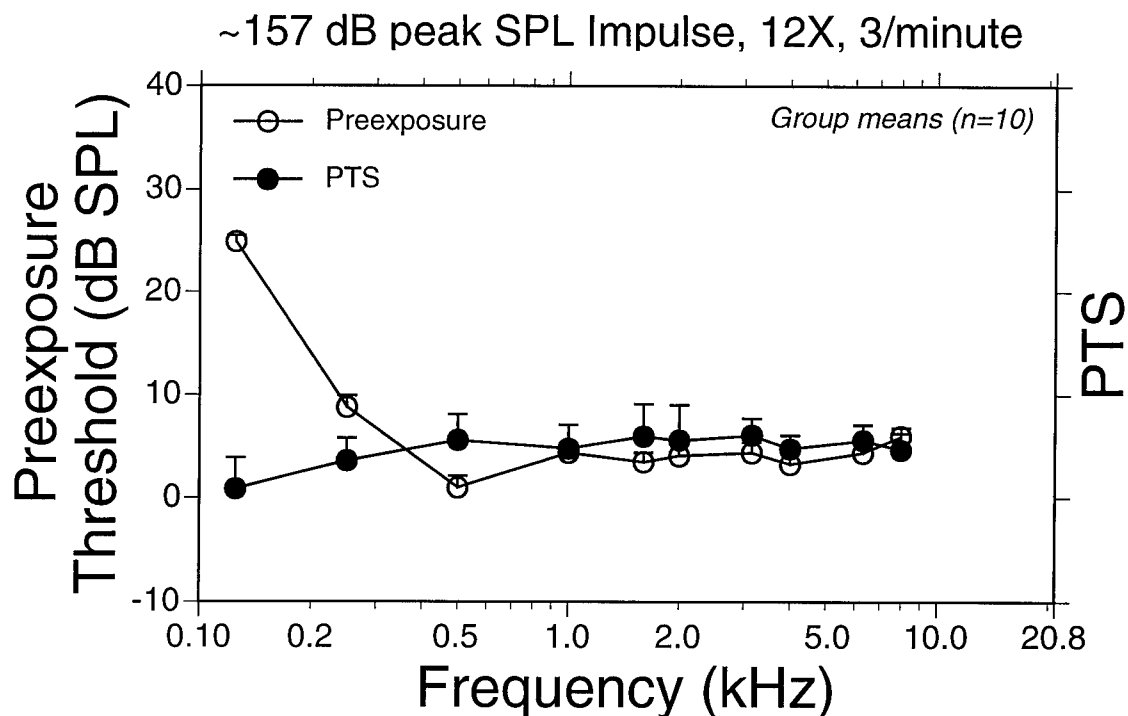
### Conventional Shock Tube

~157 dB peak SPL, 12X, 3/minute

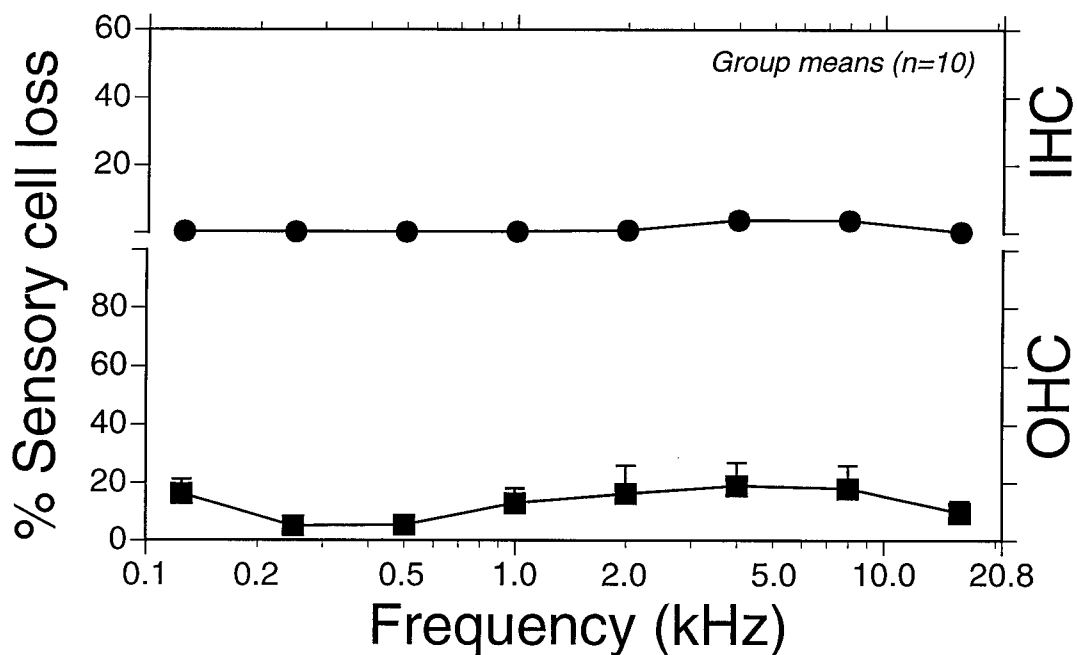
#### Animal #

A4	-	Completed the Entire Protocol
A21	-	Completed the Entire Protocol
A23	-	Completed the Entire Protocol
A38	-	Completed the Entire Protocol
A44	-	Completed the Entire Protocol
Z53	-	Completed the Entire Protocol
Z69	-	Completed the Entire Protocol
Z72	-	Completed the Entire Protocol
Z73	-	Completed the Entire Protocol
Z97	-	Completed the Entire Protocol

Includes audiometric thresholds, otoacoustic emissions,  
and histology.

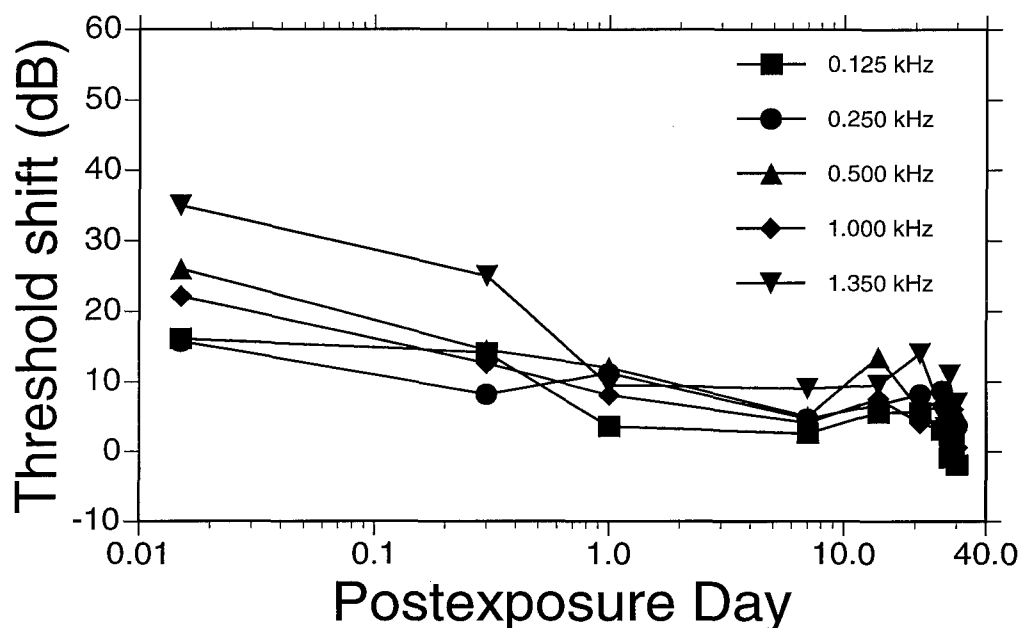


Group mean preexposure thresholds (O) and permanent threshold shifts (●) from a group of animals exposed to 12 impulses of approximately 157 dB peak SPL at a rate of 3 impulses per minute. Error bars represent one standard error of the mean.

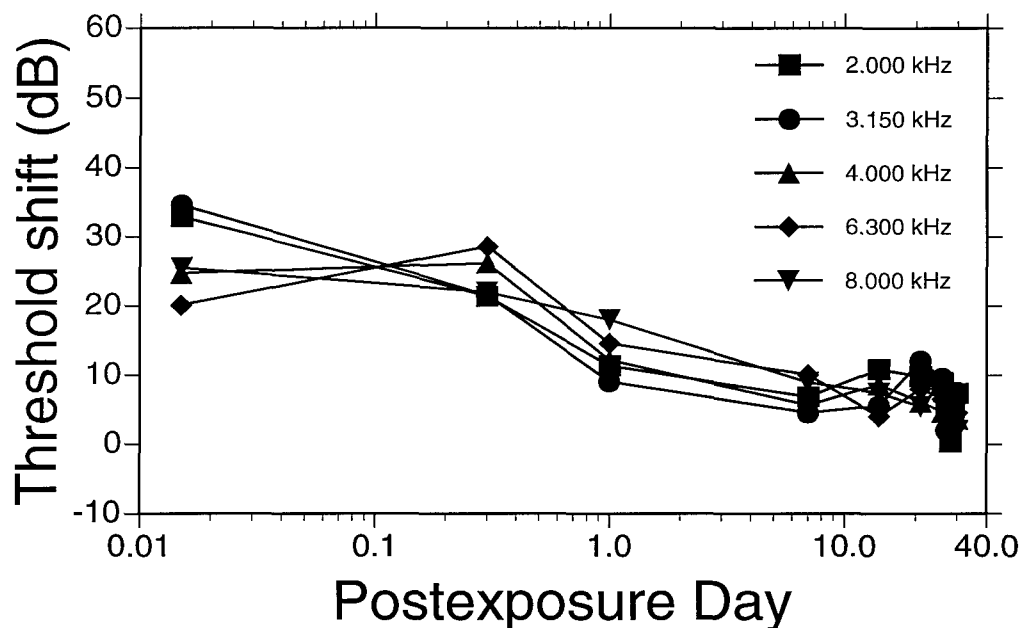


Group mean percent inner (●) and (■) outer hair cell loss from a group of animals exposed to 12 impulses of approximately 157 dB peak SPL at a rate of 3 impulses per minute. Error bars represent one standard error of the mean.

~157 dB peak SPL Impulse, 12X, 3/minute



Group mean threshold shifts at the indicated test frequencies from a group of animals exposed to 12 impulses of approximately 157 dB peak SPL at a rate of 3 impulses per minute.

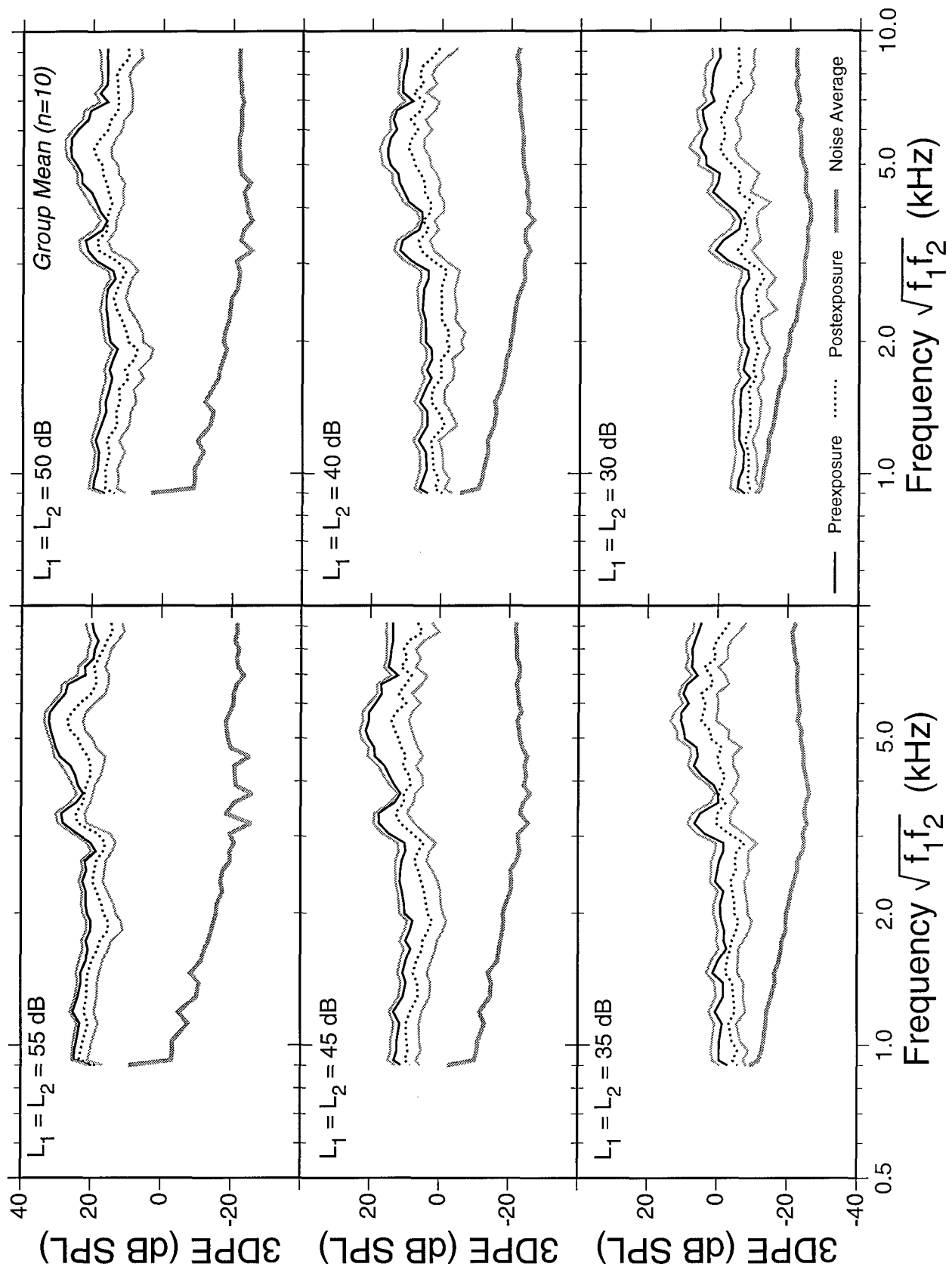


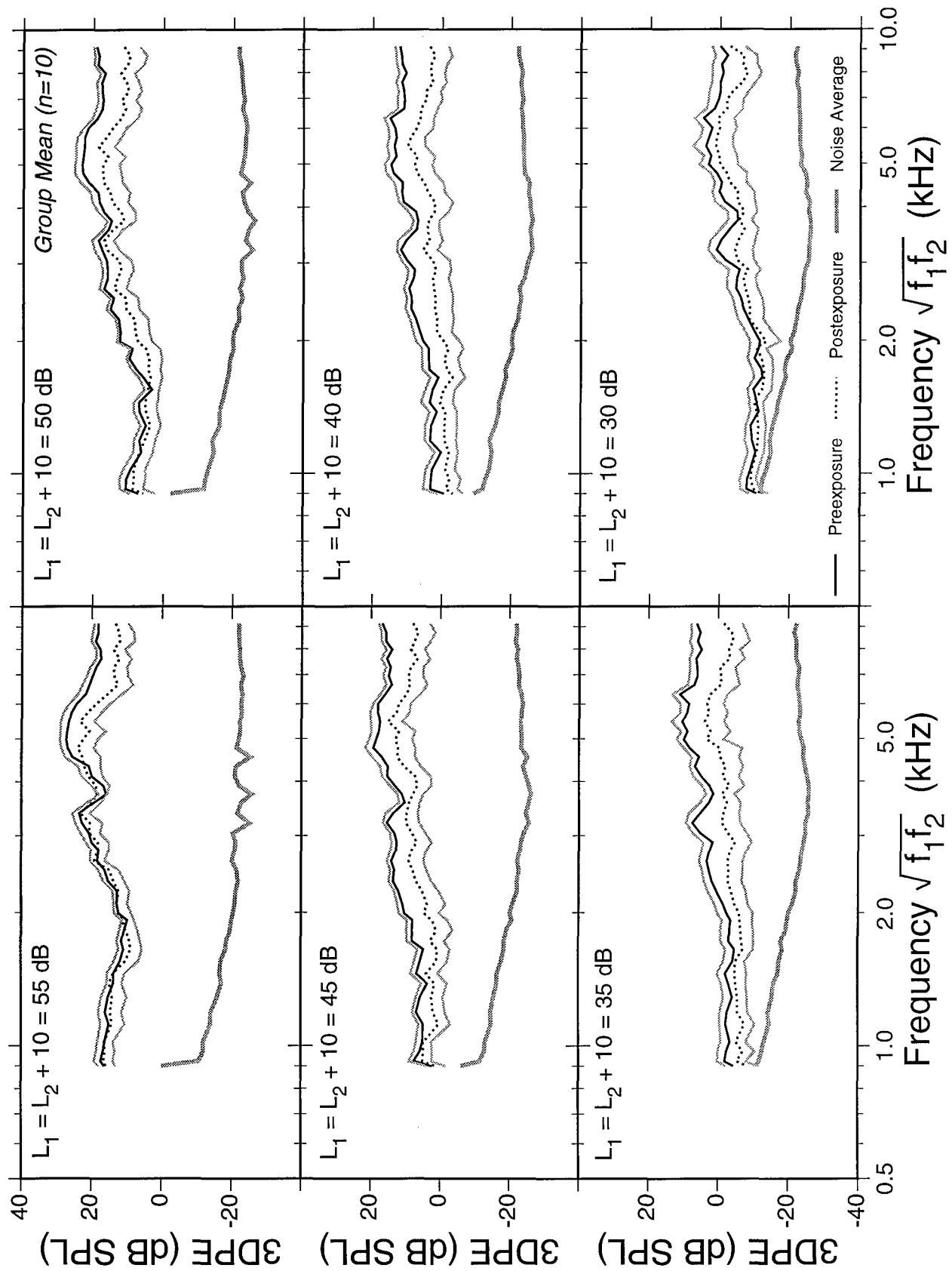
Group mean threshold shifts at the indicated test frequencies from a group of animals exposed to 12 impulses of approximately 157 dB peak SPL at a rate of 3 impulses per minute.

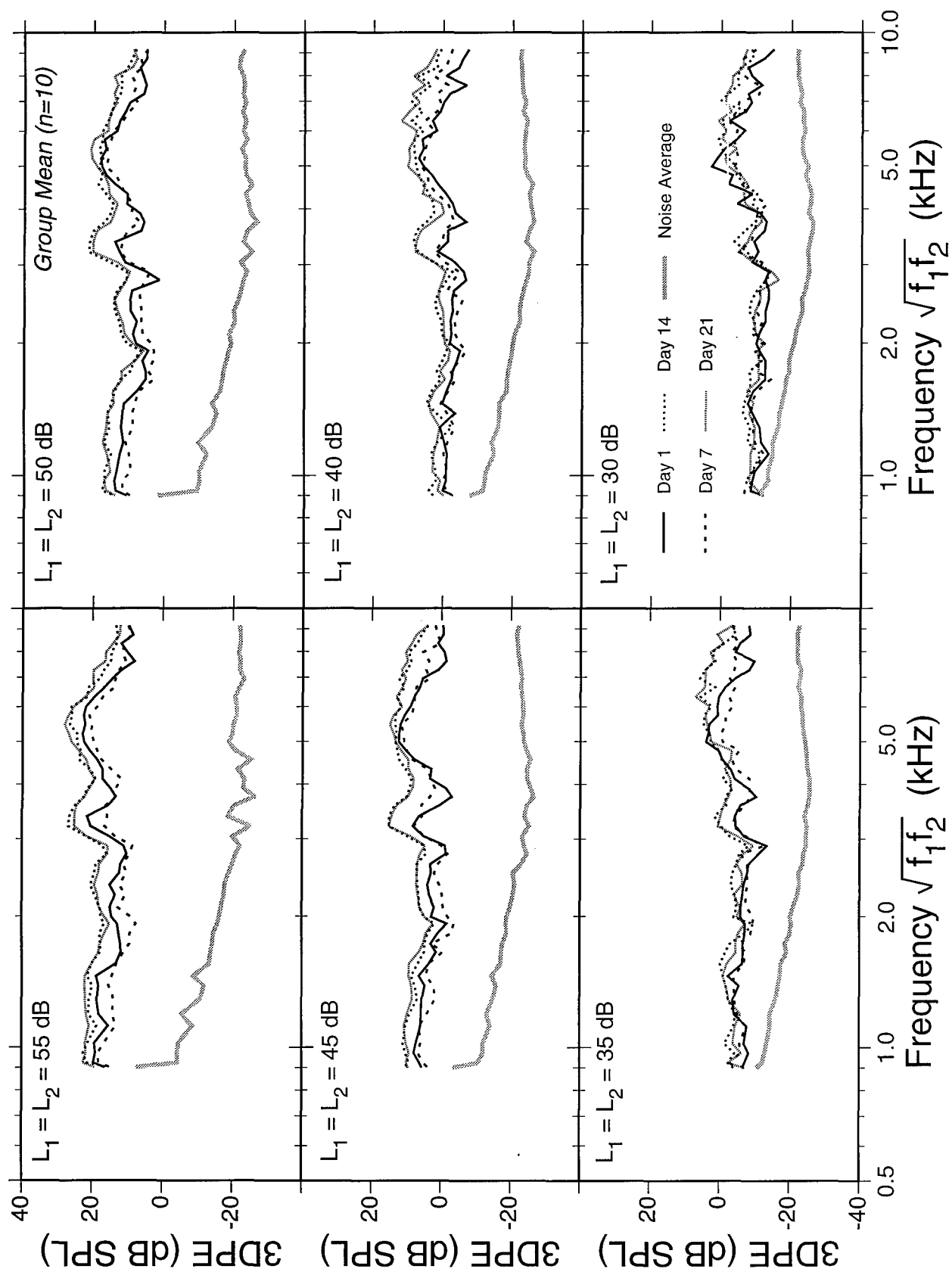
## Group Mean DPEgrams

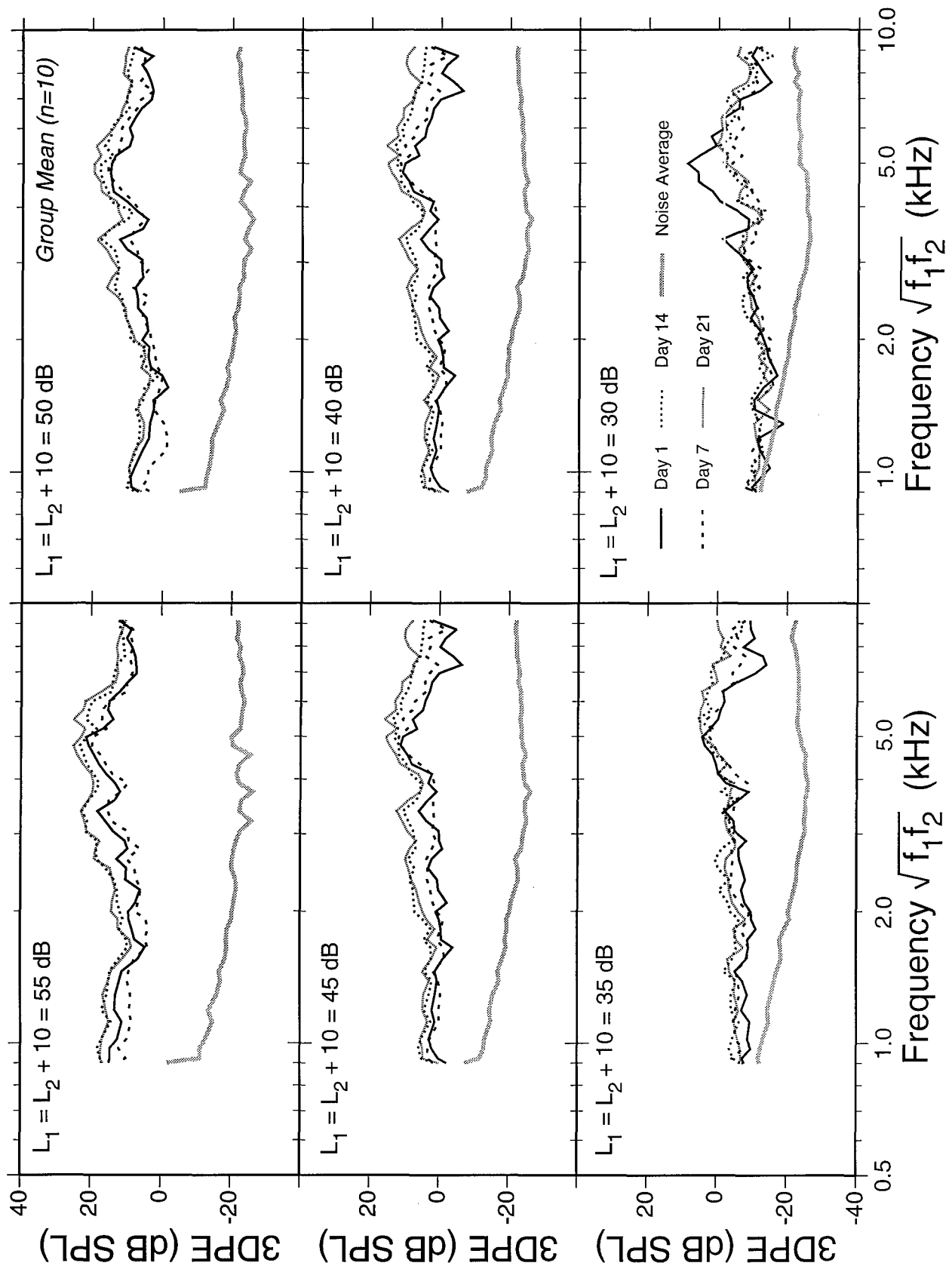
Group mean DPEgrams (Pages 72 and 73) at the indicated primary levels for a group of 10 animals exposed to 12 impulses of approximately 157 dB peak SPL at a rate of 3 impulses per minute. The solid lines represent the mean preexposure DPEgram at the six equal-primary measurements ( $L_1 = L_2 = 55$  to 30 dB SPL) and six unequal-primary ( $L_1 = L_2 + 10 = 55$  to 30 dB SPL) measurements. Each set of DPEgrams for each subject represents the average of three measurements made on different days. The dashed lines represent the group mean postexposure measurements made at least 30 days following exposure. The upper gray line represents one standard error of the mean above the preexposure measurements and the lower gray line represents one standard error of the mean below the postexposure measurements. The thick gray line represents the average noise floor over the pre- and postexposure measurements.

Group mean DPEgrams measured 1, 7, 14, and 21 days after noise exposure are presented in the next two figures (Pages 74 and 75) using the indicated equal and unequal primary levels. The solid lines represent the group mean DPEgrams measured one day following noise exposure and the various dotted lines show the group mean DPEgrams in successive tests as indicated in the legend. The thick gray line represents the average noise floor over the four postexposure measurements.











## Group Mean and Individual Audiometry

The next tables (Pages 77 through 88) present the group summary data (means and standard errors of the mean) and individual audiometric data. Preexposure measurements are the average of five threshold determinations made on different days. Postexposure measurements represent a single threshold measurement at each test frequency. The mean 30-day postexposure threshold is taken to be the average of the postexposure measurements made on Days 26 through 30. Permanent threshold shifts are computed as the 30-day postexposure threshold measure minus the preexposure threshold at each test frequency.

## Group Means

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	24.9	8.8	1.0	4.4	3.5	4.1	4.4	3.3	4.4	6.0
Postexposure Thresholds (dB SPL)										
Day 0	41.0	24.5	27.0	26.5	38.5	37.0	39.0	28.0	24.5	31.5
Day 0.3	39.0	17.0	15.5	17.0	28.5	25.5	26.0	29.5	33.0	28.0
Day 1	28.5	20.0	13.0	12.5	13.0	15.5	13.5	15.5	19.0	24.0
Day 7	27.5	13.5	6.0	8.5	12.5	11.0	9.0	9.0	14.5	15.0
Day 14	30.5	15.5	14.5	12.0	13.0	15.0	10.0	12.0	8.5	13.5
Day 21	30.5	17.0	7.0	8.5	17.5	14.0	16.5	9.5	12.5	11.5
Day 26	28.0	17.5	8.5	7.5	9.0	13.0	14.0	8.0	11.0	15.0
Day 27	28.0	12.0	4.5	11.5	7.0	10.0	6.5	8.5	10.0	13.0
Day 28	24.0	10.0	6.5	11.5	14.5	4.5	11.5	9.5	10.5	7.5
Day 29	26.0	10.0	7.0	10.5	6.5	9.5	8.5	7.5	9.5	10.0
Day 30	23.0	12.5	6.5	5.0	10.5	11.5	12.0	7.0	9.0	8.0
Postexposure Mean (26-30)	25.8	12.4	6.6	9.2	9.5	9.7	10.5	8.1	10.0	10.7

## Postexposure Threshold shifts (dB)

Day 0	16.1	15.7	26.0	22.1	35.0	32.9	34.6	24.7	20.1	25.5
Day 0.3	14.1	8.2	14.5	12.6	25.0	21.4	21.6	26.2	28.6	22.0
Day 1	3.6	11.2	12.0	8.1	9.5	11.4	9.1	12.2	14.6	18.0
Day 7	2.6	4.7	5.0	4.1	9.0	6.9	4.6	5.7	10.1	9.0
Day 14	5.6	6.7	13.5	7.6	9.5	10.9	5.6	8.7	4.1	7.5
Day 21	5.6	8.2	6.0	4.1	14.0	9.9	12.1	6.2	8.1	5.5
Day 26	3.1	8.7	7.5	3.1	5.5	8.9	9.6	4.7	6.6	9.0
Day 27	3.1	3.2	3.5	7.1	3.5	5.9	2.1	5.2	5.6	7.0
Day 28	-0.9	1.2	5.5	7.1	11.0	0.4	7.1	6.2	6.1	1.5
Day 29	1.1	1.2	6.0	6.1	3.0	5.4	4.1	4.2	5.1	4.0
Day 30	-1.9	3.7	5.5	0.6	7.0	7.4	7.6	3.7	4.6	2.0
PTS Average	0.9	3.6	5.6	4.8	6.0	5.6	6.1	4.8	5.6	4.7

## Group Standard Errors

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	0.6	1.1	1.1	0.9	0.9	1.5	1.0	1.0	0.8	0.8

## Postexposure Thresholds (dB SPL)

Day 0	3.1	3.0	4.6	5.3	7.8	7.1	7.2	7.0	5.7	5.6
Day 0.3	5.1	4.8	3.6	3.9	5.4	4.7	4.4	5.1	6.2	4.5
Day 1	5.9	3.5	5.6	4.8	2.7	3.5	6.6	6.8	5.4	5.3
Day 7	2.6	3.1	2.1	2.7	3.5	3.9	3.4	4.5	5.3	3.8
Day 14	3.1	2.3	5.1	4.8	5.6	3.0	1.7	2.5	2.4	1.6
Day 21	2.3	2.5	3.0	1.6	3.7	2.9	2.7	3.3	2.8	2.8
Day 26	2.9	2.0	2.9	1.7	1.7	3.3	1.7	1.9	2.9	3.6
Day 27	2.2	2.3	1.9	3.1	2.9	4.2	2.2	2.8	2.1	2.7
Day 28	4.7	3.5	3.5	2.8	4.0	2.4	2.6	4.2	2.1	2.1
Day 29	4.2	3.4	4.9	4.6	3.9	3.9	2.1	3.3	1.9	1.5
Day 30	4.8	3.2	3.2	2.5	2.8	3.1	1.9	2.0	2.8	2.2
Postexposure Mean (26-30)	3.1	1.9	2.2	1.7	2.4	2.4	0.9	1.4	1.4	1.2

## Postexposure Threshold shifts (dB)

Day 0	3.3	3.7	4.5	5.2	7.5	7.8	7.1	7.3	5.9	5.9
Day 0.3	5.2	5.0	3.8	4.3	5.3	4.9	4.8	5.5	6.2	4.6
Day 1	6.0	4.2	5.9	4.7	3.0	4.3	6.8	6.6	5.3	5.4
Day 7	2.8	3.5	2.2	3.1	3.7	4.4	3.7	4.6	5.4	3.6
Day 14	3.2	2.4	5.1	4.6	5.5	3.7	2.0	2.7	2.9	2.3
Day 21	2.3	3.0	3.0	2.0	3.8	2.8	2.9	3.7	2.4	3.0
Day 26	3.1	2.3	3.5	2.3	2.4	4.2	1.8	1.6	2.8	3.5
Day 27	1.9	2.2	1.6	3.4	3.8	4.9	2.6	2.3	2.1	3.1
Day 28	4.4	3.7	3.4	3.2	4.3	3.2	3.2	4.2	2.3	2.3
Day 29	4.1	3.8	5.3	4.8	4.5	4.5	2.0	3.3	2.0	1.4
Day 30	4.9	3.5	3.2	2.9	3.0	3.6	2.6	2.3	3.1	2.7
PTS Average	3.0	2.2	2.5	2.3	3.1	3.4	1.6	1.3	1.5	1.6

## Subject A4

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	23.5	5.5	4.5	4.5	2.5	6.5	6.5	-0.5	5.5	7.5
Postexposure Thresholds (dB SPL)										
Day 0	57.5	32.5	57.5	52.5	67.5	67.5	72.5	62.5	22.5	52.5
Day 0.3	67.5	-7.5	27.5	42.5	67.5	12.5	22.5	37.5	57.5	47.5
Day 1	22.5	22.5	12.5	37.5	32.5	17.5	-7.5	32.5	17.5	27.5
Day 7	27.5	12.5	7.5	12.5	22.5	7.5	22.5	12.5	47.5	22.5
Day 14	47.5	2.5	12.5	17.5	12.5	7.5	2.5	7.5	7.5	12.5
Day 21	42.5	32.5	7.5	7.5	7.5	22.5	27.5	17.5	22.5	22.5
Day 26	32.5	12.5	7.5	2.5	7.5	2.5	12.5	12.5	12.5	17.5
Day 27	27.5	12.5	12.5	2.5	2.5	12.5	2.5	7.5	12.5	22.5
Day 28	27.5	12.5	7.5	7.5	12.5	7.5	7.5	12.5	17.5	22.5
Day 29	32.5	7.5	12.5	12.5	2.5	12.5	7.5	7.5	7.5	7.5
Day 30	27.5	17.5	7.5	2.5	7.5	2.5	12.5	7.5	17.5	12.5
Postexposure Mean (26-30)	29.5	12.5	9.5	5.5	6.5	7.5	8.5	9.5	13.5	16.5
Postexposure Threshold shifts (dB)										
Day 0	34.0	27.0	53.0	48.0	65.0	61.0	66.0	63.0	17.0	45.0
Day 0.3	44.0	-13.0	23.0	38.0	65.0	6.0	16.0	38.0	52.0	40.0
Day 1	-1.0	17.0	8.0	33.0	30.0	11.0	-14.0	33.0	12.0	20.0
Day 7	4.0	7.0	3.0	8.0	20.0	1.0	16.0	13.0	42.0	15.0
Day 14	24.0	-3.0	8.0	13.0	10.0	1.0	-4.0	8.0	2.0	5.0
Day 21	19.0	27.0	3.0	3.0	5.0	16.0	21.0	18.0	17.0	15.0
Day 26	9.0	7.0	3.0	-2.0	5.0	-4.0	6.0	13.0	7.0	10.0
Day 27	4.0	7.0	8.0	-2.0	0.0	6.0	-4.0	8.0	7.0	15.0
Day 28	4.0	7.0	3.0	3.0	10.0	1.0	1.0	13.0	12.0	15.0
Day 29	9.0	2.0	8.0	8.0	0.0	6.0	1.0	8.0	2.0	0.0
Day 30	4.0	12.0	3.0	-2.0	5.0	-4.0	6.0	8.0	12.0	5.0
PTS Average	6.0	7.0	5.0	1.0	4.0	1.0	2.0	10.0	8.0	9.0

## Subject A21

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	23.5	8.5	3.5	7.5	5.5	7.5	5.5	4.5	4.5	4.5
Postexposure Thresholds (dB SPL)										
Day 0	32.5	12.5	12.5	7.5	7.5	12.5	2.5	-7.5	7.5	7.5
Day 0.3	27.5	27.5	12.5	12.5	17.5	12.5	7.5	7.5	7.5	12.5
Day 1	12.5	12.5	12.5	22.5	12.5	12.5	-2.5	-7.5	2.5	27.5
Day 7	27.5	17.5	7.5	-7.5	12.5	7.5	2.5	-7.5	2.5	17.5
Day 14	37.5	12.5	57.5	52.5	62.5	32.5	7.5	2.5	7.5	7.5
Day 21	27.5	17.5	2.5	2.5	32.5	12.5	7.5	2.5	7.5	2.5
Day 26	32.5	17.5	-7.5	2.5	7.5	7.5	12.5	7.5	2.5	-7.5
Day 27	27.5	12.5	7.5	7.5	2.5	22.5	17.5	7.5	12.5	7.5
Day 28	22.5	12.5	2.5	12.5	2.5	7.5	2.5	12.5	-2.5	2.5
Day 29	32.5	12.5	-7.5	12.5	2.5	2.5	7.5	-7.5	12.5	7.5
Day 30	22.5	7.5	2.5	12.5	12.5	12.5	17.5	12.5	2.5	7.5
Postexposure Mean (26-30)	27.5	12.5	-0.5	9.5	5.5	10.5	11.5	6.5	5.5	3.5

## Postexposure Threshold shifts (dB)

Day 0	9.0	4.0	9.0	0.0	2.0	5.0	-3.0	-12.0	3.0	3.0
Day 0.3	4.0	19.0	9.0	5.0	12.0	5.0	2.0	3.0	3.0	8.0
Day 1	-11.0	4.0	9.0	15.0	7.0	5.0	-8.0	-12.0	-2.0	23.0
Day 7	4.0	9.0	4.0	-15.0	7.0	0.0	-3.0	-12.0	-2.0	13.0
Day 14	14.0	4.0	54.0	45.0	57.0	25.0	2.0	-2.0	3.0	3.0
Day 21	4.0	9.0	-1.0	-5.0	27.0	5.0	2.0	-2.0	3.0	-2.0
Day 26	9.0	9.0	-11.0	-5.0	2.0	0.0	7.0	3.0	-2.0	-12.0
Day 27	4.0	4.0	4.0	0.0	-3.0	15.0	12.0	3.0	8.0	3.0
Day 28	-1.0	4.0	-1.0	5.0	-3.0	0.0	-3.0	8.0	-7.0	-2.0
Day 29	9.0	4.0	-11.0	5.0	-3.0	-5.0	2.0	-12.0	8.0	3.0
Day 30	-1.0	-1.0	-1.0	5.0	7.0	5.0	12.0	8.0	-2.0	3.0
PTS Average	4.0	4.0	-4.0	2.0	0.0	3.0	6.0	2.0	1.0	-1.0

## Subject A23

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	26.5	7.5	0.5	8.5	7.5	-2.5	-0.5	-0.5	5.5	7.5
Postexposure Thresholds (dB SPL)										
Day 0	42.5	22.5	32.5	32.5	42.5	47.5	22.5	27.5	22.5	17.5
Day 0.3	32.5	12.5	12.5	12.5	22.5	22.5	27.5	22.5	12.5	17.5
Day 1	22.5	12.5	7.5	-7.5	12.5	12.5	2.5	7.5	12.5	12.5
Day 7	27.5	2.5	-2.5	2.5	7.5	-7.5	2.5	2.5	7.5	7.5
Day 14	32.5	7.5	12.5	7.5	2.5	12.5	2.5	7.5	12.5	12.5
Day 21	37.5	12.5	2.5	7.5	7.5	22.5	12.5	12.5	7.5	22.5
Day 26	27.5	12.5	7.5	7.5	7.5	17.5	7.5	2.5	7.5	12.5
Day 27	22.5	7.5	2.5	12.5	2.5	2.5	7.5	7.5	7.5	12.5
Day 28	27.5	12.5	7.5	12.5	27.5	7.5	17.5	32.5	17.5	12.5
Day 29	42.5	17.5	2.5	2.5	2.5	2.5	7.5	2.5	12.5	12.5
Day 30	22.5	17.5	7.5	7.5	17.5	7.5	12.5	2.5	7.5	12.5
Postexposure Mean (26-30)	28.5	13.5	5.5	8.5	11.5	7.5	10.5	9.5	10.5	12.5
Postexposure Threshold shifts (dB)										
Day 0	16.0	15.0	32.0	24.0	35.0	50.0	23.0	28.0	17.0	10.0
Day 0.3	6.0	5.0	12.0	4.0	15.0	25.0	28.0	23.0	7.0	10.0
Day 1	-4.0	5.0	7.0	-16.0	5.0	15.0	3.0	8.0	7.0	5.0
Day 7	1.0	-5.0	-3.0	-6.0	0.0	-5.0	3.0	3.0	2.0	0.0
Day 14	6.0	0.0	12.0	-1.0	-5.0	15.0	3.0	8.0	7.0	5.0
Day 21	11.0	5.0	2.0	-1.0	0.0	25.0	13.0	13.0	2.0	15.0
Day 26	1.0	5.0	7.0	-1.0	0.0	20.0	8.0	3.0	2.0	5.0
Day 27	-4.0	0.0	2.0	4.0	-5.0	5.0	8.0	8.0	2.0	5.0
Day 28	1.0	5.0	7.0	4.0	20.0	10.0	18.0	33.0	12.0	5.0
Day 29	16.0	10.0	2.0	-6.0	-5.0	5.0	8.0	3.0	7.0	5.0
Day 30	-4.0	10.0	7.0	-1.0	10.0	10.0	13.0	3.0	2.0	5.0
PTS Average	2.0	6.0	5.0	0.0	4.0	10.0	11.0	10.0	5.0	5.0

## Subject A38

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	23.5	10.5	0.5	8.5	7.5	5.5	6.5	5.5	7.5	9.5

## Postexposure Thresholds (dB SPL)

Day 0	52.5	32.5	42.5	52.5	82.5	7.5	67.5	2.5	-7.5	17.5
Day 0.3	12.5	32.5	-7.5	2.5	27.5	22.5	57.5	42.5	57.5	7.5
Day 1	62.5	12.5	7.5	32.5	2.5	32.5	57.5	37.5	62.5	62.5
Day 7	17.5	22.5	2.5	17.5	27.5	27.5	2.5	32.5	32.5	32.5
Day 14	22.5	22.5	2.5	7.5	7.5	22.5	17.5	27.5	12.5	7.5
Day 21	22.5	12.5	2.5	7.5	27.5	27.5	27.5	-12.5	32.5	-7.5
Day 26	22.5	22.5	2.5	2.5	2.5	22.5	22.5	12.5	32.5	37.5
Day 27	32.5	17.5	12.5	22.5	-7.5	7.5	-7.5	27.5	22.5	2.5
Day 28	2.5	-17.5	-12.5	12.5	17.5	-7.5	2.5	-2.5	12.5	2.5
Day 29	7.5	-7.5	-17.5	-2.5	-12.5	-7.5	2.5	2.5	12.5	17.5
Day 30	2.5	-7.5	-7.5	-7.5	7.5	12.5	2.5	-7.5	7.5	-7.5
Postexposure Mean (26-30)	13.5	1.5	-4.5	5.5	1.5	5.5	4.5	6.5	17.5	10.5

## Postexposure Threshold shifts (dB)

Day 0	29.0	22.0	42.0	44.0	75.0	2.0	61.0	-3.0	-15.0	8.0
Day 0.3	-11.0	22.0	-8.0	-6.0	20.0	17.0	51.0	37.0	50.0	-2.0
Day 1	39.0	2.0	7.0	24.0	-5.0	27.0	51.0	32.0	55.0	53.0
Day 7	-6.0	12.0	2.0	9.0	20.0	22.0	-4.0	27.0	25.0	23.0
Day 14	-1.0	12.0	2.0	-1.0	0.0	17.0	11.0	22.0	5.0	-2.0
Day 21	-1.0	2.0	2.0	-1.0	20.0	22.0	21.0	-18.0	25.0	-17.0
Day 26	-1.0	12.0	2.0	-6.0	-5.0	17.0	16.0	7.0	25.0	28.0
Day 27	9.0	7.0	12.0	14.0	-15.0	2.0	-14.0	22.0	15.0	-7.0
Day 28	-21.0	-28.0	-13.0	4.0	10.0	-13.0	-4.0	-8.0	5.0	-7.0
Day 29	-16.0	-18.0	-18.0	-11.0	-20.0	-13.0	-4.0	-3.0	5.0	8.0
Day 30	-21.0	-18.0	-8.0	-16.0	0.0	7.0	-4.0	-13.0	0.0	-17.0
PTS Average	-10.0	-9.0	-5.0	-3.0	-6.0	0.0	-2.0	1.0	10.0	1.0

## Subject A44

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	27.5	4.5	-4.5	1.5	4.5	7.5	8.5	5.5	7.5	3.5
Postexposure Thresholds (dB SPL)										
Day 0	32.5	22.5	22.5	27.5	57.5	47.5	52.5	52.5	52.5	57.5
Day 0.3	32.5	12.5	12.5	7.5	27.5	32.5	12.5	47.5	22.5	37.5
Day 1	2.5	27.5	12.5	12.5	7.5	12.5	12.5	27.5	17.5	12.5
Day 7	12.5	12.5	-2.5	7.5	2.5	-2.5	2.5	-2.5	2.5	12.5
Day 14	22.5	17.5	12.5	12.5	7.5	12.5	12.5	12.5	-12.5	17.5
Day 21	22.5	7.5	2.5	17.5	2.5	7.5	7.5	12.5	12.5	12.5
Day 26	12.5	12.5	22.5	7.5	2.5	2.5	12.5	7.5	7.5	17.5
Day 27	27.5	17.5	2.5	12.5	7.5	2.5	2.5	7.5	12.5	22.5
Day 28	12.5	2.5	7.5	17.5	-7.5	-7.5	12.5	2.5	7.5	7.5
Day 29	12.5	-2.5	22.5	-7.5	-2.5	12.5	22.5	12.5	17.5	2.5
Day 30	-7.5	2.5	-7.5	2.5	-7.5	-7.5	7.5	12.5	-7.5	2.5
Postexposure Mean (26-30)	11.5	6.5	9.5	6.5	-1.5	0.5	11.5	8.5	7.5	10.5

## Postexposure Threshold shifts (dB)

Day 0	5.0	18.0	27.0	26.0	53.0	40.0	44.0	47.0	45.0	54.0
Day 0.3	5.0	8.0	17.0	6.0	23.0	25.0	4.0	42.0	15.0	34.0
Day 1	-25.0	23.0	17.0	11.0	3.0	5.0	4.0	22.0	10.0	9.0
Day 7	-15.0	8.0	2.0	6.0	-2.0	-10.0	-6.0	-8.0	-5.0	9.0
Day 14	-5.0	13.0	17.0	11.0	3.0	5.0	4.0	7.0	-20.0	14.0
Day 21	-5.0	3.0	7.0	16.0	-2.0	0.0	-1.0	7.0	5.0	9.0
Day 26	-15.0	8.0	27.0	6.0	-2.0	-5.0	4.0	2.0	0.0	14.0
Day 27	0.0	13.0	7.0	11.0	3.0	-5.0	-6.0	2.0	5.0	19.0
Day 28	-15.0	-2.0	12.0	16.0	-12.0	-15.0	4.0	-3.0	0.0	4.0
Day 29	-15.0	-7.0	27.0	-9.0	-7.0	5.0	14.0	7.0	10.0	-1.0
Day 30	-35.0	-2.0	-3.0	1.0	-12.0	-15.0	-1.0	7.0	-15.0	-1.0
PTS Average	-16.0	2.0	14.0	5.0	-6.0	-7.0	3.0	3.0	0.0	7.0



## Subject Z53

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	23.5	8.5	-2.5	5.5	4.5	3.5	3.5	0.5	1.5	2.5

## Postexposure Thresholds (dB SPL)

Day 0	47.5	27.5	17.5	12.5	32.5	57.5	47.5	47.5	12.5	47.5
Day 0.3	57.5	22.5	12.5	17.5	47.5	57.5	22.5	57.5	32.5	42.5
Day 1	22.5	12.5	2.5	7.5	22.5	7.5	-7.5	-7.5	27.5	37.5
Day 7	27.5	2.5	2.5	7.5	-7.5	2.5	-7.5	7.5	12.5	-7.5
Day 14	17.5	22.5	17.5	2.5	7.5	7.5	7.5	7.5	12.5	17.5
Day 21	22.5	12.5	2.5	12.5	22.5	7.5	17.5	-2.5	2.5	12.5
Day 26	22.5	22.5	7.5	2.5	7.5	2.5	7.5	2.5	2.5	17.5
Day 27	22.5	-2.5	2.5	-7.5	2.5	-7.5	2.5	2.5	2.5	12.5
Day 28	12.5	7.5	2.5	-7.5	17.5	7.5	12.5	-7.5	12.5	12.5
Day 29	12.5	7.5	17.5	22.5	17.5	2.5	7.5	7.5	12.5	12.5
Day 30	22.5	12.5	7.5	2.5	12.5	12.5	17.5	2.5	7.5	12.5
Postexposure Mean (26-30)	18.5	9.5	7.5	2.5	11.5	3.5	9.5	1.5	7.5	13.5

## Postexposure Threshold shifts (dB)

Day 0	24.0	19.0	20.0	7.0	28.0	54.0	44.0	47.0	11.0	45.0
Day 0.3	34.0	14.0	15.0	12.0	43.0	54.0	19.0	57.0	31.0	40.0
Day 1	-1.0	4.0	5.0	2.0	18.0	4.0	-11.0	-8.0	26.0	35.0
Day 7	4.0	-6.0	5.0	2.0	-12.0	-1.0	-11.0	7.0	11.0	-10.0
Day 14	-6.0	14.0	20.0	-3.0	3.0	4.0	4.0	7.0	11.0	15.0
Day 21	-1.0	4.0	5.0	7.0	18.0	4.0	14.0	-3.0	1.0	10.0
Day 26	-1.0	14.0	10.0	-3.0	3.0	-1.0	4.0	2.0	1.0	15.0
Day 27	-1.0	-11.0	5.0	-13.0	-2.0	-11.0	-1.0	2.0	1.0	10.0
Day 28	-11.0	-1.0	5.0	-13.0	13.0	4.0	9.0	-8.0	11.0	10.0
Day 29	-11.0	-1.0	20.0	17.0	13.0	-1.0	4.0	7.0	11.0	10.0
Day 30	-1.0	4.0	10.0	-3.0	8.0	9.0	14.0	2.0	6.0	10.0
PTS Average	-5.0	1.0	10.0	-3.0	7.0	0.0	6.0	1.0	6.0	11.0

## Subject Z69

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	24.5	4.5	-0.5	1.5	1.5	-4.5	-1.5	4.5	1.5	10.5
Postexposure Thresholds (dB SPL)										
Day 0	42.5	42.5	32.5	37.5	37.5	62.5	47.5	22.5	47.5	32.5
Day 0.3	52.5	37.5	32.5	32.5	27.5	17.5	37.5	27.5	62.5	42.5
Day 1	57.5	47.5	62.5	17.5	7.5	37.5	37.5	52.5	12.5	2.5
Day 7	42.5	32.5	17.5	22.5	27.5	32.5	17.5	12.5	7.5	12.5
Day 14	42.5	22.5	12.5	2.5	7.5	22.5	17.5	17.5	7.5	7.5
Day 21	32.5	27.5	2.5	2.5	27.5	2.5	17.5	17.5	7.5	12.5
Day 26	47.5	27.5	12.5	7.5	17.5	32.5	17.5	17.5	12.5	7.5
Day 27	32.5	7.5	2.5	12.5	12.5	32.5	7.5	7.5	7.5	12.5
Day 28	32.5	22.5	7.5	27.5	27.5	7.5	17.5	2.5	7.5	2.5
Day 29	47.5	32.5	32.5	37.5	22.5	37.5	2.5	32.5	7.5	12.5
Day 30	42.5	27.5	22.5	22.5	27.5	22.5	22.5	12.5	2.5	2.5
Postexposure Mean (26-30)	40.5	23.5	15.5	21.5	21.5	26.5	13.5	14.5	7.5	7.5
Postexposure Threshold shifts (dB)										
Day 0	18.0	38.0	33.0	36.0	36.0	67.0	49.0	18.0	46.0	22.0
Day 0.3	28.0	33.0	33.0	31.0	26.0	22.0	39.0	23.0	61.0	32.0
Day 1	33.0	43.0	63.0	16.0	6.0	42.0	39.0	48.0	11.0	-8.0
Day 7	18.0	28.0	18.0	21.0	26.0	37.0	19.0	8.0	6.0	2.0
Day 14	18.0	18.0	13.0	1.0	6.0	27.0	19.0	13.0	6.0	-3.0
Day 21	8.0	23.0	3.0	1.0	26.0	7.0	19.0	13.0	6.0	2.0
Day 26	23.0	23.0	13.0	6.0	16.0	37.0	19.0	13.0	11.0	-3.0
Day 27	8.0	3.0	3.0	11.0	11.0	37.0	9.0	3.0	6.0	2.0
Day 28	8.0	18.0	8.0	26.0	26.0	12.0	19.0	-2.0	6.0	-8.0
Day 29	23.0	28.0	33.0	36.0	21.0	42.0	4.0	28.0	6.0	2.0
Day 30	18.0	23.0	23.0	21.0	26.0	27.0	24.0	8.0	1.0	-8.0
PTS Average	16.0	19.0	16.0	20.0	20.0	31.0	15.0	10.0	6.0	-3.0

## Subject Z72

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	25.5	13.5	6.5	1.5	1.5	0.5	6.5	7.5	3.5	5.5
Postexposure Thresholds (dB SPL)										
Day 0	42.5	22.5	17.5	12.5	12.5	22.5	32.5	27.5	37.5	42.5
Day 0.3	32.5	22.5	12.5	22.5	17.5	22.5	17.5	17.5	22.5	17.5
Day 1	27.5	22.5	2.5	7.5	7.5	7.5	12.5	12.5	2.5	12.5
Day 7	27.5	2.5	2.5	7.5	7.5	12.5	7.5	-7.5	-2.5	7.5
Day 14	32.5	12.5	2.5	2.5	7.5	22.5	7.5	7.5	12.5	12.5
Day 21	27.5	12.5	2.5	2.5	2.5	2.5	7.5	12.5	7.5	12.5
Day 26	27.5	22.5	2.5	12.5	7.5	7.5	12.5	7.5	2.5	12.5
Day 27	27.5	22.5	7.5	27.5	12.5	22.5	7.5	12.5	2.5	7.5
Day 28	22.5	17.5	7.5	12.5	2.5	12.5	12.5	22.5	2.5	7.5
Day 29	22.5	7.5	2.5	12.5	2.5	2.5	7.5	2.5	2.5	2.5
Day 30	27.5	12.5	2.5	2.5	7.5	12.5	12.5	7.5	22.5	12.5
Postexposure Mean (26-30)	25.5	16.5	4.5	13.5	6.5	11.5	10.5	10.5	6.5	8.5
Postexposure Threshold shifts (dB)										
Day 0	17.0	9.0	11.0	11.0	11.0	22.0	26.0	20.0	34.0	37.0
Day 0.3	7.0	9.0	6.0	21.0	16.0	22.0	11.0	10.0	19.0	12.0
Day 1	2.0	9.0	-4.0	6.0	6.0	7.0	6.0	5.0	-1.0	7.0
Day 7	2.0	-11.0	-4.0	6.0	6.0	12.0	1.0	-15.0	-6.0	2.0
Day 14	7.0	-1.0	-4.0	1.0	6.0	22.0	1.0	0.0	9.0	7.0
Day 21	2.0	-1.0	-4.0	1.0	1.0	2.0	1.0	5.0	4.0	7.0
Day 26	2.0	9.0	-4.0	11.0	6.0	7.0	6.0	0.0	-1.0	7.0
Day 27	2.0	9.0	1.0	26.0	11.0	22.0	1.0	5.0	-1.0	2.0
Day 28	-3.0	4.0	1.0	11.0	1.0	12.0	6.0	15.0	-1.0	2.0
Day 29	-3.0	-6.0	-4.0	11.0	1.0	2.0	1.0	-5.0	-1.0	-3.0
Day 30	2.0	-1.0	-4.0	1.0	6.0	12.0	6.0	0.0	19.0	7.0
PTS Average	0.0	3.0	-2.0	12.0	5.0	11.0	4.0	3.0	3.0	3.0

## Subject Z73

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	28.5	12.5	3.5	3.5	-1.5	9.5	3.5	6.5	0.5	3.5
Postexposure Thresholds (dB SPL)										
Day 0	32.5	12.5	22.5	12.5	32.5	32.5	32.5	32.5	27.5	27.5
Day 0.3	37.5	-7.5	12.5	7.5	7.5	12.5	27.5	12.5	22.5	22.5
Day 1	32.5	12.5	2.5	-7.5	12.5	12.5	22.5	12.5	22.5	22.5
Day 7	32.5	12.5	12.5	2.5	12.5	17.5	27.5	32.5	32.5	32.5
Day 14	27.5	12.5	2.5	2.5	12.5	2.5	12.5	7.5	12.5	17.5
Day 21	37.5	12.5	32.5	12.5	27.5	12.5	27.5	12.5	12.5	12.5
Day 26	32.5	7.5	22.5	17.5	12.5	22.5	12.5	12.5	17.5	22.5
Day 27	42.5	17.5	2.5	7.5	27.5	12.5	12.5	12.5	17.5	27.5
Day 28	57.5	17.5	32.5	12.5	32.5	12.5	27.5	22.5	17.5	2.5
Day 29	27.5	12.5	12.5	22.5	27.5	17.5	17.5	12.5	12.5	12.5
Day 30	37.5	22.5	22.5	2.5	12.5	27.5	7.5	7.5	17.5	12.5
Postexposure Mean (26-30)	39.5	15.5	18.5	12.5	22.5	18.5	15.5	13.5	16.5	15.5

## Postexposure Threshold shifts (dB)

Day 0	4.0	0.0	19.0	9.0	34.0	23.0	29.0	26.0	27.0	24.0
Day 0.3	9.0	-20.0	9.0	4.0	9.0	3.0	24.0	6.0	22.0	19.0
Day 1	4.0	0.0	-1.0	-11.0	14.0	3.0	19.0	6.0	22.0	19.0
Day 7	4.0	0.0	9.0	-1.0	14.0	8.0	24.0	26.0	32.0	29.0
Day 14	-1.0	0.0	-1.0	-1.0	14.0	-7.0	9.0	1.0	12.0	14.0
Day 21	9.0	0.0	29.0	9.0	29.0	3.0	24.0	6.0	12.0	9.0
Day 26	4.0	-5.0	19.0	14.0	14.0	13.0	9.0	6.0	17.0	19.0
Day 27	14.0	5.0	-1.0	4.0	29.0	3.0	9.0	6.0	17.0	24.0
Day 28	29.0	5.0	29.0	9.0	34.0	3.0	24.0	16.0	17.0	-1.0
Day 29	-1.0	0.0	9.0	19.0	29.0	8.0	14.0	6.0	12.0	9.0
Day 30	9.0	10.0	19.0	-1.0	14.0	18.0	4.0	1.0	17.0	9.0
PTS Average	11.0	3.0	15.0	9.0	24.0	9.0	12.0	7.0	16.0	12.0

## Subject Z97

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Preexposure Threshold (dB SPL)	22.5	12.5	-1.5	1.5	1.5	7.5	5.5	-0.5	6.5	5.5
Postexposure Thresholds (dB SPL)										
Day 0	27.5	17.5	12.5	17.5	12.5	12.5	12.5	12.5	22.5	12.5
Day 0.3	37.5	17.5	27.5	12.5	22.5	42.5	27.5	22.5	32.5	32.5
Day 1	22.5	17.5	7.5	2.5	12.5	2.5	7.5	-12.5	12.5	22.5
Day 7	32.5	17.5	12.5	12.5	12.5	12.5	12.5	7.5	2.5	12.5
Day 14	22.5	22.5	12.5	12.5	2.5	7.5	12.5	22.5	12.5	22.5
Day 21	32.5	22.5	12.5	12.5	17.5	22.5	12.5	22.5	12.5	12.5
Day 26	22.5	17.5	7.5	12.5	17.5	12.5	22.5	-2.5	12.5	12.5
Day 27	17.5	7.5	-7.5	17.5	7.5	-7.5	12.5	-7.5	2.5	2.5
Day 28	22.5	12.5	2.5	7.5	12.5	-2.5	2.5	-2.5	12.5	2.5
Day 29	22.5	12.5	-7.5	-7.5	2.5	12.5	2.5	2.5	-2.5	12.5
Day 30	32.5	12.5	7.5	2.5	7.5	12.5	7.5	12.5	12.5	12.5
Postexposure Mean (26-30)	23.5	12.5	0.5	6.5	9.5	5.5	9.5	0.5	7.5	8.5
Postexposure Threshold shifts (dB)										
Day 0	5.0	5.0	14.0	16.0	11.0	5.0	7.0	13.0	16.0	7.0
Day 0.3	15.0	5.0	29.0	11.0	21.0	35.0	22.0	23.0	26.0	27.0
Day 1	0.0	5.0	9.0	1.0	11.0	-5.0	2.0	-12.0	6.0	17.0
Day 7	10.0	5.0	14.0	11.0	11.0	5.0	7.0	8.0	-4.0	7.0
Day 14	0.0	10.0	14.0	11.0	1.0	0.0	7.0	23.0	6.0	17.0
Day 21	10.0	10.0	14.0	11.0	16.0	15.0	7.0	23.0	6.0	7.0
Day 26	0.0	5.0	9.0	11.0	16.0	5.0	17.0	-2.0	6.0	7.0
Day 27	-5.0	-5.0	-6.0	16.0	6.0	-15.0	7.0	-7.0	-4.0	-3.0
Day 28	0.0	0.0	4.0	6.0	11.0	-10.0	-3.0	-2.0	6.0	-3.0
Day 29	0.0	0.0	-6.0	-9.0	1.0	5.0	-3.0	3.0	-9.0	7.0
Day 30	10.0	0.0	9.0	1.0	6.0	5.0	2.0	13.0	6.0	7.0
PTS Average	1.0	0.0	2.0	5.0	8.0	-2.0	4.0	1.0	1.0	3.0

## Individual and Group Mean Histology Summary

Group summary data (means, standard deviations, and standard errors of the mean) are presented for total and percent cell losses measured in octave-band lengths of the cochlea on Pages 90 and 91. Following the summary data, individual animal total and percent cell losses are presented (Pages 92 through 98). Following the tabulated data, individual animal cochleograms are presented on Pages 99 through 108. The three graphs on these pages show: (top) a "standard" cochleogram showing percent inner and outer sensory cell losses with the permanent threshold shift (dB); (middle) percent cell losses in each of the three rows of outer hair cells; and (bottom) percent missing supporting (pillar) cells.

~157 dB peak SPL Impulses, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Group means							
0.125 kHz	0.6	15.6	31.9	42.9	90.4	0.1	0.7
0.25 kHz	0.7	6.3	11.9	31.5	49.7	0.0	0.2
0.5 kHz	0.6	10.2	22.8	19.2	52.2	0.4	0.2
1 kHz	0.8	38.4	51.3	30.6	120.3	0.1	0.4
2 kHz	1.8	53.2	53.7	50.0	156.9	0.9	2.5
4 kHz	9.4	58.6	65.6	60.1	184.3	20.8	14.5
8 kHz	9.0	49.2	61.1	59.4	169.7	15.6	13.7
16 kHz	0.5	22.9	26.8	31.9	81.6	0.6	1.8
TOTALS	23.4	254.4	325.1	325.6	905.1	38.5	34.0

Group standard deviations

0.125 kHz	0.7	19.4	35.1	37.6	91.0	0.3	1.6
0.25 kHz	1.3	3.7	8.9	29.1	33.9	0.0	0.4
0.5 kHz	0.7	11.6	27.6	19.1	51.6	1.3	0.6
1 kHz	0.9	64.9	65.7	34.8	149.1	0.3	0.8
2 kHz	4.0	106.7	102.8	90.9	300.0	2.5	6.9
4 kHz	14.5	81.5	89.6	76.4	244.0	33.4	22.8
8 kHz	11.0	65.4	87.1	84.7	236.3	20.1	16.0
16 kHz	0.8	29.2	37.4	44.6	109.7	1.1	4.1
TOTALS	14.9	290.1	339.6	284.1	905.0	33.5	27.2

Group standard errors

0.125 kHz	0.2	6.1	11.1	11.9	28.8	0.1	0.5
0.25 kHz	0.4	1.2	2.8	9.2	10.7	0.0	0.1
0.5 kHz	0.2	3.7	8.7	6.1	16.3	0.4	0.2
1 kHz	0.3	20.5	20.8	11.0	47.1	0.1	0.3
2 kHz	1.3	33.8	32.5	28.7	94.9	0.8	2.2
4 kHz	4.6	25.8	28.3	24.2	77.2	10.6	7.2
8 kHz	3.5	20.7	27.5	26.8	74.7	6.3	5.1
16 kHz	0.3	9.2	11.8	14.1	34.7	0.3	1.3
TOTALS	4.7	91.7	107.4	89.8	286.2	10.6	8.6

~157 dB peak SPL Impulses, 12X, 3/minute

Percent sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Group means							
0.125 kHz	0.42	8.33	17.16	23.33	16.27	0.04	0.38
0.25 kHz	0.31	1.98	3.74	9.68	5.13	0.00	0.06
0.5 kHz	0.25	3.19	7.17	6.03	5.46	0.09	0.07
1 kHz	0.34	12.14	16.61	10.23	12.99	0.02	0.12
2 kHz	0.76	16.41	16.69	15.54	16.21	0.17	0.76
4 kHz	3.73	18.03	20.27	18.62	18.97	3.90	4.39
8 kHz	3.69	15.75	19.49	18.96	18.07	3.16	4.47
16 kHz	0.24	8.14	9.58	11.33	9.68	0.13	0.63

Group standard deviations

0.125 kHz	0.48	10.16	18.30	19.56	15.79	0.13	0.88
0.25 kHz	0.58	1.22	2.80	8.74	3.38	0.00	0.13
0.5 kHz	0.29	3.55	8.82	5.92	5.44	0.28	0.22
1 kHz	0.37	20.22	20.73	12.28	15.73	0.06	0.25
2 kHz	1.63	32.49	31.21	27.53	30.36	0.47	2.10
4 kHz	5.69	24.92	27.62	23.67	25.05	6.27	6.96
8 kHz	4.44	20.35	27.22	26.39	24.55	4.04	5.07
16 kHz	0.41	10.13	13.02	15.53	12.71	0.24	1.41

Group standard errors

0.125 kHz	0.15	3.21	5.79	6.19	4.99	0.04	0.28
0.25 kHz	0.18	0.39	0.88	2.76	1.07	0.00	0.04
0.5 kHz	0.09	1.12	2.79	1.87	1.72	0.09	0.07
1 kHz	0.12	6.39	6.56	3.88	4.97	0.02	0.08
2 kHz	0.52	10.27	9.87	8.70	9.60	0.15	0.66
4 kHz	1.80	7.88	8.73	7.49	7.92	1.98	2.20
8 kHz	1.40	6.43	8.61	8.34	7.76	1.28	1.60
16 kHz	0.13	3.20	4.12	4.91	4.02	0.08	0.45



~157 dB peak SPL Impulses, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla A4							
0.125 kHz	1	60	110	126	296	0	0
0.25 kHz	0	5	28	83	116	0	0
0.5 kHz	0	38	65	26	129	0	0
1 kHz	1	34	49	27	110	0	0
2 kHz	0	8	9	12	29	0	0
4 kHz	2	93	116	126	335	4	1
8 kHz	20	166	177	190	533	34	45
16 kHz	0	88	114	114	316	0	13
TOTALS	24	492	668	704	1864	38	59
Chinchilla A21							
0.125 kHz	0	3	6	19	28	0	0
0.25 kHz	1	3	6	9	18	0	0
0.5 kHz	1	2	2	6	10	0	0
1 kHz	0	2	2	2	6	0	0
2 kHz	2	0	5	7	12	0	0
4 kHz	1	1	3	3	7	0	0
8 kHz	10	24	19	24	67	22	12
16 kHz	0	8	21	29	58	1	0
TOTALS	15	43	64	99	206	23	12
Chinchilla A23							
0.125 kHz	1	27	53	71	151	1	5
0.25 kHz	0	6	19	63	88	0	0
0.5 kHz	1	3	18	31	52	0	0
1 kHz	1	5	31	27	63	0	0
2 kHz	1	11	26	20	57	1	1
4 kHz	0	1	4	5	10	0	0
8 kHz	14	30	29	31	90	12	17
16 kHz	1	6	0	0	6	0	0
TOTALS	19	89	180	248	517	14	23

~157 dB peak SPL Impulses, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla A38							
0.125 kHz	0	3	12	15	30	0	1
0.25 kHz	0	3	4	13	20	0	0
0.5 kHz	0	4	10	9	23	0	0
1 kHz	1	111	109	51	271	1	2
2 kHz	0	148	135	106	389	0	2
4 kHz	43	159	216	209	584	78	53
8 kHz	1	173	260	241	674	1	10
16 kHz	2	62	69	113	244	2	3
TOTALS	47	663	815	757	2235	82	71
Chinchilla A44							
0.125 kHz	2	10	6	7	23	0	1
0.25 kHz	0	11	7	9	27	0	1
0.5 kHz	1	4	4	1	9	0	0
1 kHz	0	11	14	9	34	0	0
2 kHz	0	7	3	7	17	0	0
4 kHz	28	52	46	48	146	78	41
8 kHz	0	2	2	4	8	0	0
16 kHz	0	11	15	26	52	0	0
TOTALS	31	108	97	111	316	78	43
Chinchilla Z53							
0.125 kHz	1	4	15	19	38	0	0
0.25 kHz	4	6	12	16	34	0	0
0.5 kHz	2	6	2	6	14	0	0
1 kHz	0	10	4	12	26	0	0
2 kHz	1	20	9	23	52	0	0
4 kHz	4	6	8	10	24	0	0
8 kHz	34	45	52	44	141	62	38
16 kHz	0	18	15	12	45	3	2
TOTALS	46	115	117	142	374	65	40

~157 dB peak SPL Impulses, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla Z69							
0.125 kHz	0	36	72	75	183	0	0
0.25 kHz	0	3	15	66	84	0	1
0.5 kHz	0	6	48	57	111	0	0
1 kHz	3	197	205	56	458	0	2
2 kHz	13	329	323	294	946	8	22
4 kHz	10	238	229	161	628	47	48
8 kHz	1	5	14	2	21	20	0
16 kHz	0	3	0	0	3	0	0
TOTALS	27	817	906	711	2434	75	73
Chinchilla Z72							
0.125 kHz	1	5	16	27	48	0	0
0.25 kHz	2	10	22	7	39	0	0
0.5 kHz	0	19	71	43	133	4	2
1 kHz	1	8	87	113	208	0	0
2 kHz	0	7	21	20	48	0	0
4 kHz	3	8	15	12	35	0	0
8 kHz	7	41	50	45	136	5	13
16 kHz	2	27	34	25	86	0	0
TOTALS	16	125	316	292	733	9	15
Chinchilla Z73							
0.125 kHz	0	1	21	49	71	0	0
0.25 kHz	0	3	1	40	44	0	0
0.5 kHz	1	18	2	9	29	0	0
1 kHz	0	3	5	6	14	0	0
2 kHz	0	2	3	6	11	0	0
4 kHz	1	26	18	24	68	1	2
8 kHz	1	6	7	11	24	0	2
16 kHz	0	6	0	0	6	0	0
TOTALS	3	65	57	145	267	1	4

~157 dB peak SPL Impulses, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner	1st row	2nd row	3rd row	Comb.	Inner	Outer
	hair	outer	outer	outer	outer	pillar	pillar
	cells	hair	hair	hair	hair	cells	cells
	cells	cells	cells	cells	cells	cells	cells
Chinchilla Z97							
0.125 kHz	0	7	8	21	36	0	0
0.25 kHz	0	13	5	9	27	0	0
0.5 kHz	0	2	6	4	12	0	0
1 kHz	1	3	7	3	13	0	0
2 kHz	1	0	3	5	8	0	0
4 kHz	2	2	1	3	6	0	0
8 kHz	2	0	1	2	3	0	0
16 kHz	0	0	0	0	0	0	0
TOTALS	6	27	31	47	105	0	0

~157 dB peak SPL Impulses, 12X, 3/minute

Percent sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla A4							
0.125 kHz	0.7	31.4	57.6	66.0	51.7	0.0	0.0
0.25 kHz	0.0	1.5	8.4	24.9	11.6	0.0	0.0
0.5 kHz	0.0	11.4	19.5	7.8	12.9	0.0	0.0
1 kHz	0.4	10.7	15.5	8.5	11.6	0.0	0.0
2 kHz	0.0	2.5	2.8	3.7	3.0	0.0	0.0
4 kHz	0.8	28.6	35.7	38.8	34.4	0.8	0.3
8 kHz	7.7	51.2	54.6	58.6	54.8	6.5	13.9
16 kHz	0.0	30.3	39.3	39.3	36.3	0.0	4.5
Chinchilla A21							
0.125 kHz	0.0	1.8	3.7	11.6	5.7	0.0	0.0
0.25 kHz	0.5	1.0	2.1	3.1	2.1	0.0	0.0
0.5 kHz	0.5	0.7	0.7	2.1	1.2	0.0	0.0
1 kHz	0.0	0.7	0.7	0.7	0.7	0.0	0.0
2 kHz	1.0	0.0	1.8	2.5	1.4	0.0	0.0
4 kHz	0.5	0.4	1.1	1.1	0.9	0.0	0.0
8 kHz	4.5	8.6	6.8	8.6	8.0	4.9	4.3
16 kHz	0.0	3.2	8.4	11.6	7.7	0.2	0.0
Chinchilla A23							
0.125 kHz	0.7	15.2	29.8	39.9	28.3	0.4	2.8
0.25 kHz	0.0	1.9	6.1	20.1	9.4	0.0	0.0
0.5 kHz	0.4	1.0	5.8	9.9	5.6	0.0	0.0
1 kHz	0.4	1.7	10.4	9.1	7.1	0.0	0.0
2 kHz	0.4	3.6	8.6	6.6	6.3	0.2	0.3
4 kHz	0.0	0.3	1.3	1.7	1.1	0.0	0.0
8 kHz	5.7	9.9	9.6	10.3	9.9	2.5	5.6
16 kHz	0.5	2.2	0.0	0.0	0.7	0.0	0.0
Chinchilla A38							
0.125 kHz	0.0	1.6	6.5	8.1	5.4	0.0	0.5
0.25 kHz	0.0	0.9	1.2	4.0	2.0	0.0	0.0
0.5 kHz	0.0	1.2	3.1	2.8	2.4	0.0	0.0
1 kHz	0.4	35.8	35.2	16.5	29.2	0.2	0.6
2 kHz	0.0	46.7	42.6	33.4	40.9	0.0	0.6
4 kHz	17.4	50.2	68.1	65.9	61.4	15.3	16.7
8 kHz	0.4	54.6	82.0	76.0	70.9	0.2	3.2
16 kHz	0.9	21.9	24.4	39.9	28.7	0.4	1.1

~157 dB peak SPL Impulses, 12X, 3/minute

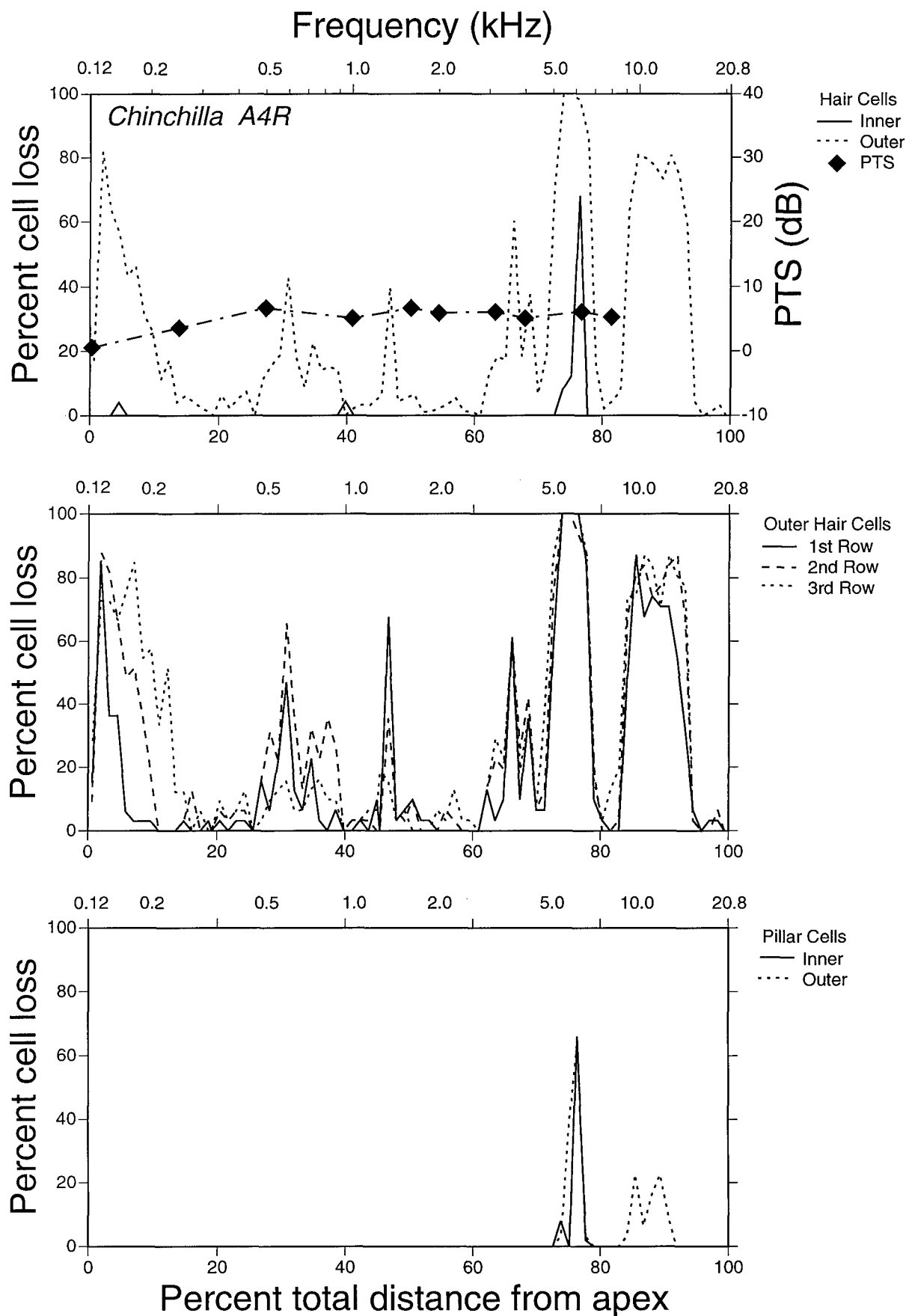
Percent sensory cell losses over octave band frequencies

		1st row	2nd row	3rd row	Comb.		
	Inner	outer	outer	outer	outer	Inner	Outer
	hair	hair	hair	hair	hair	pillar	pillar
	cells	cells	cells	cells	cells	cells	cells
Chinchilla A44							
0.125 kHz	1.3	4.9	2.9	3.4	3.7	0.0	0.5
0.25 kHz	0.0	3.1	1.9	2.5	2.5	0.0	0.3
0.5 kHz	0.4	1.1	1.1	0.3	0.8	0.0	0.0
1 kHz	0.0	3.2	4.1	2.6	3.3	0.0	0.0
2 kHz	0.0	2.0	0.9	2.0	1.6	0.0	0.0
4 kHz	10.3	14.9	13.2	13.8	14.0	13.9	11.8
8 kHz	0.0	0.6	0.6	1.1	0.8	0.0	0.0
16 kHz	0.0	3.5	4.8	8.3	5.5	0.0	0.0
Chinchilla Z53							
0.125 kHz	0.7	2.2	8.3	10.6	7.0	0.0	0.0
0.25 kHz	1.7	1.9	3.8	5.1	3.6	0.0	0.0
0.5 kHz	0.8	1.9	0.6	1.9	1.5	0.0	0.0
1 kHz	0.0	3.3	1.3	4.0	2.9	0.0	0.0
2 kHz	0.4	6.5	2.9	7.5	5.6	0.0	0.0
4 kHz	1.7	2.0	2.6	3.3	2.6	0.0	0.0
8 kHz	13.8	14.8	17.0	14.4	15.4	12.6	12.5
16 kHz	0.0	6.6	5.5	4.4	5.5	0.7	0.7
Chinchilla Z69							
0.125 kHz	0.0	18.6	37.1	38.7	31.5	0.0	0.0
0.25 kHz	0.0	0.9	4.4	19.4	8.2	0.0	0.3
0.5 kHz	0.0	1.8	14.1	16.7	10.9	0.0	0.0
1 kHz	1.2	61.0	63.5	17.3	47.3	0.0	0.6
2 kHz	5.3	99.7	97.9	89.1	95.6	1.5	6.7
4 kHz	3.9	72.1	69.4	48.8	63.4	8.8	14.5
8 kHz	0.4	1.5	4.2	0.6	2.1	3.8	0.0
16 kHz	0.0	1.0	0.0	0.0	0.3	0.0	0.0
Chinchilla Z72							
0.125 kHz	0.8	3.0	9.6	16.2	9.6	0.0	0.0
0.25 kHz	0.9	3.4	7.5	2.4	4.4	0.0	0.0
0.5 kHz	0.0	6.5	24.2	14.7	15.1	0.9	0.7
1 kHz	0.5	2.9	31.3	40.6	24.9	0.0	0.0
2 kHz	0.0	2.5	7.4	7.0	5.6	0.0	0.0
4 kHz	1.4	2.8	5.3	4.2	4.1	0.0	0.0
8 kHz	3.1	14.4	17.6	15.8	15.9	1.1	4.6
16 kHz	1.0	10.6	13.4	9.8	11.3	0.0	0.0

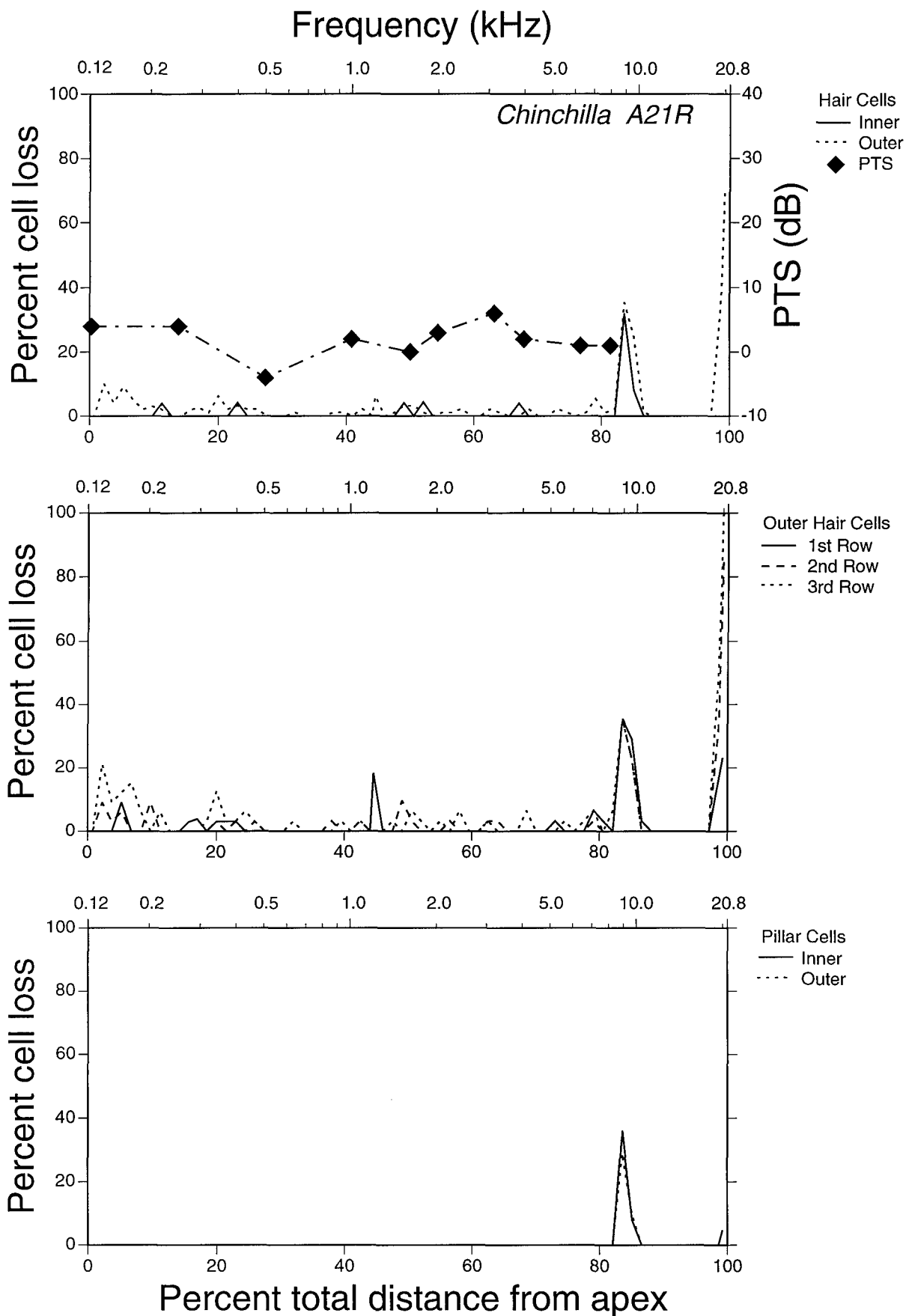
~157 dB peak SPL Impulses, 12X, 3/minute

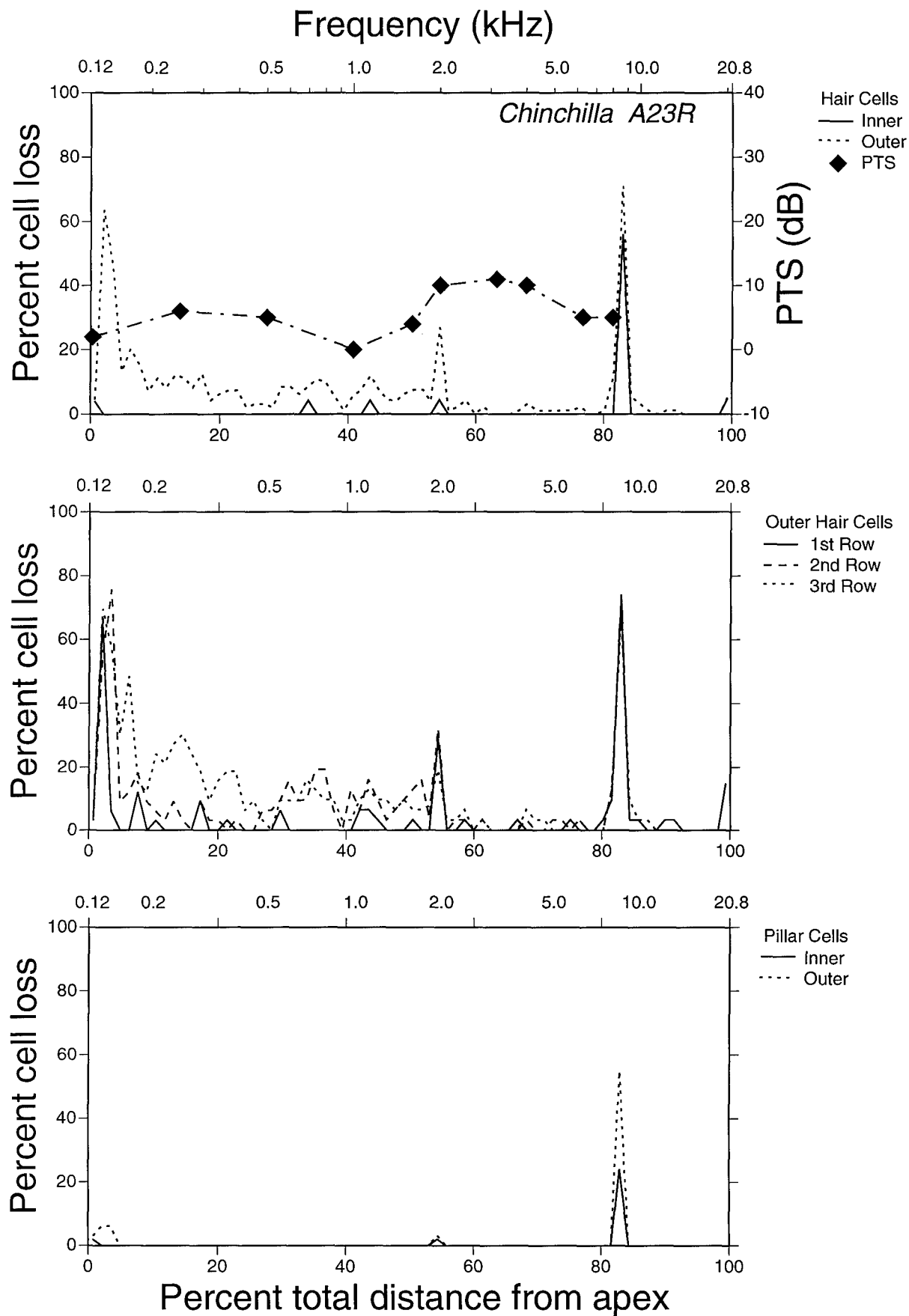
Percent sensory cell losses over octave band frequencies

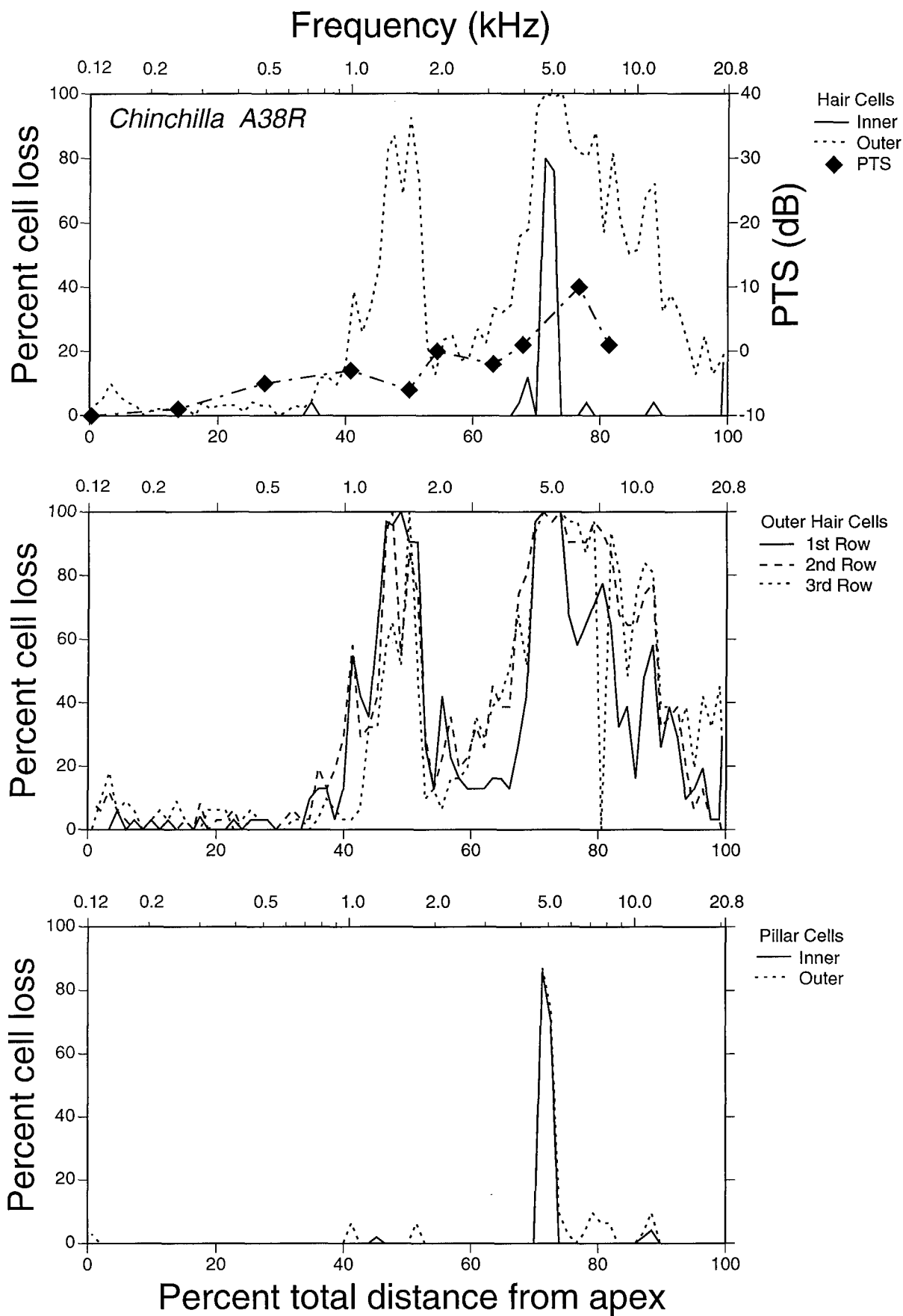
		1st row	2nd row	3rd row	Comb.		
	Inner	outer	outer	outer	outer	Inner	Outer
	hair	hair	hair	hair	hair	pillar	pillar
	cells	cells	cells	cells	cells	cells	cells
Chinchilla Z73							
0.125 kHz	0.0	0.5	11.4	26.5	12.8	0.0	0.0
0.25 kHz	0.0	0.9	0.3	12.3	4.5	0.0	0.0
0.5 kHz	0.4	5.6	0.6	2.8	3.0	0.0	0.0
1 kHz	0.0	1.0	1.6	1.9	1.5	0.0	0.0
2 kHz	0.0	0.6	1.0	1.9	1.2	0.0	0.0
4 kHz	0.4	8.3	5.7	7.6	7.2	0.2	0.6
8 kHz	0.4	1.9	2.2	3.5	2.5	0.0	0.6
16 kHz	0.0	2.1	0.0	0.0	0.7	0.0	0.0
Chinchilla Z97							
0.125 kHz	0.0	4.1	4.7	12.3	7.0	0.0	0.0
0.25 kHz	0.0	4.3	1.7	3.0	3.0	0.0	0.0
0.5 kHz	0.0	0.7	2.0	1.3	1.3	0.0	0.0
1 kHz	0.5	1.1	2.5	1.1	1.6	0.0	0.0
2 kHz	0.5	0.0	1.0	1.7	0.9	0.0	0.0
4 kHz	0.9	0.7	0.3	1.0	0.7	0.0	0.0
8 kHz	0.9	0.0	0.3	0.7	0.3	0.0	0.0
16 kHz	0.0	0.0	0.0	0.0	0.0	0.0	0.0

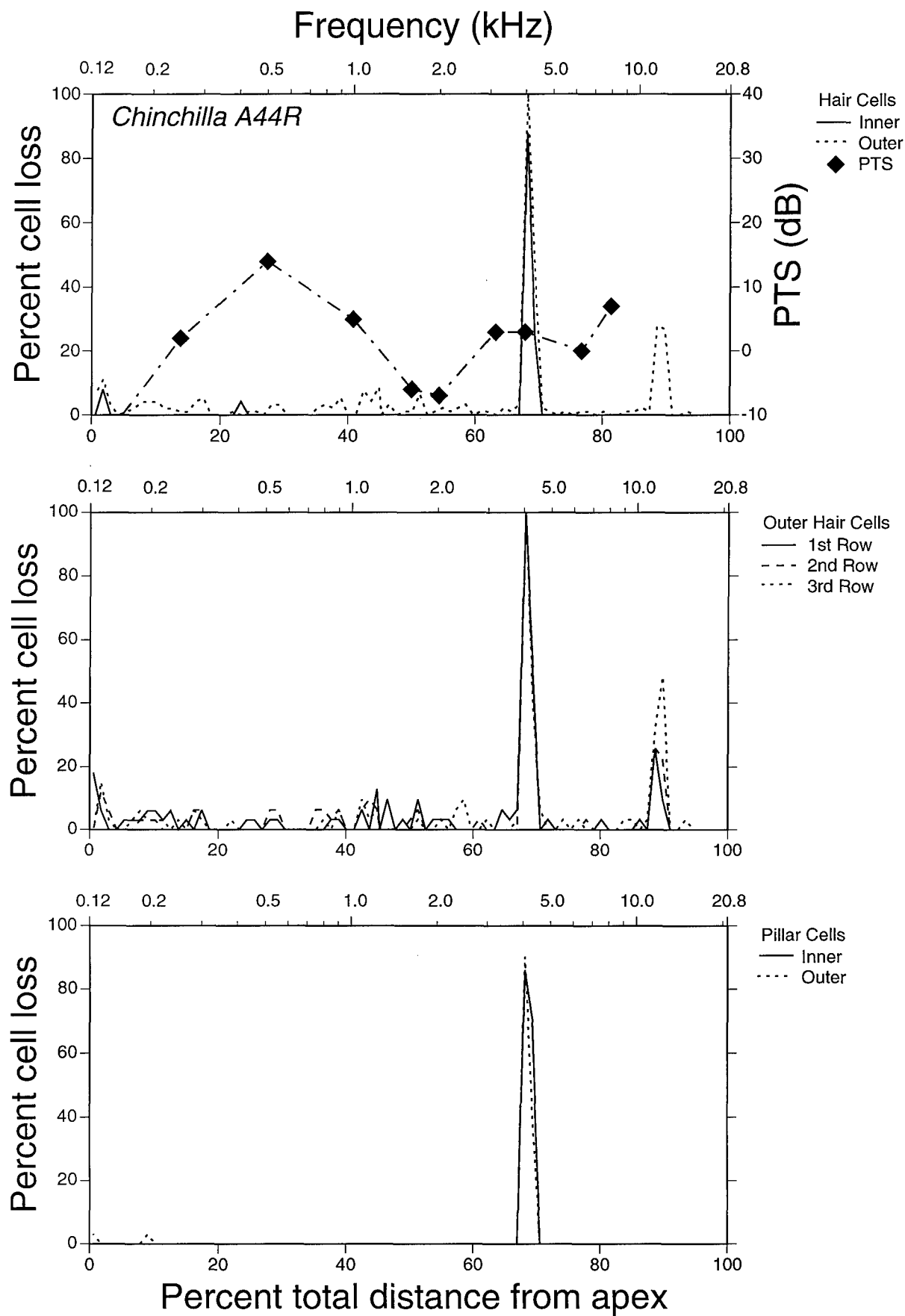


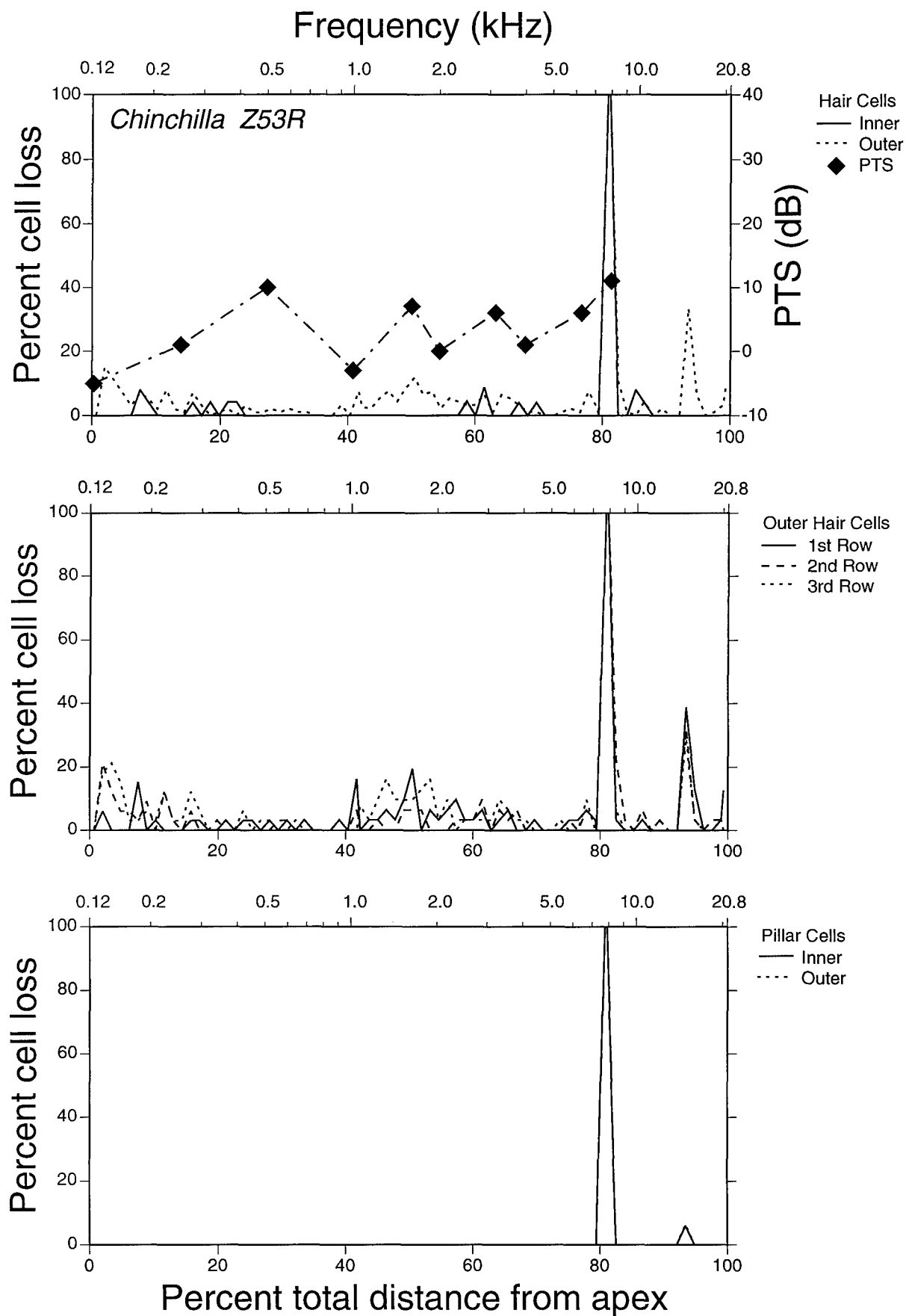


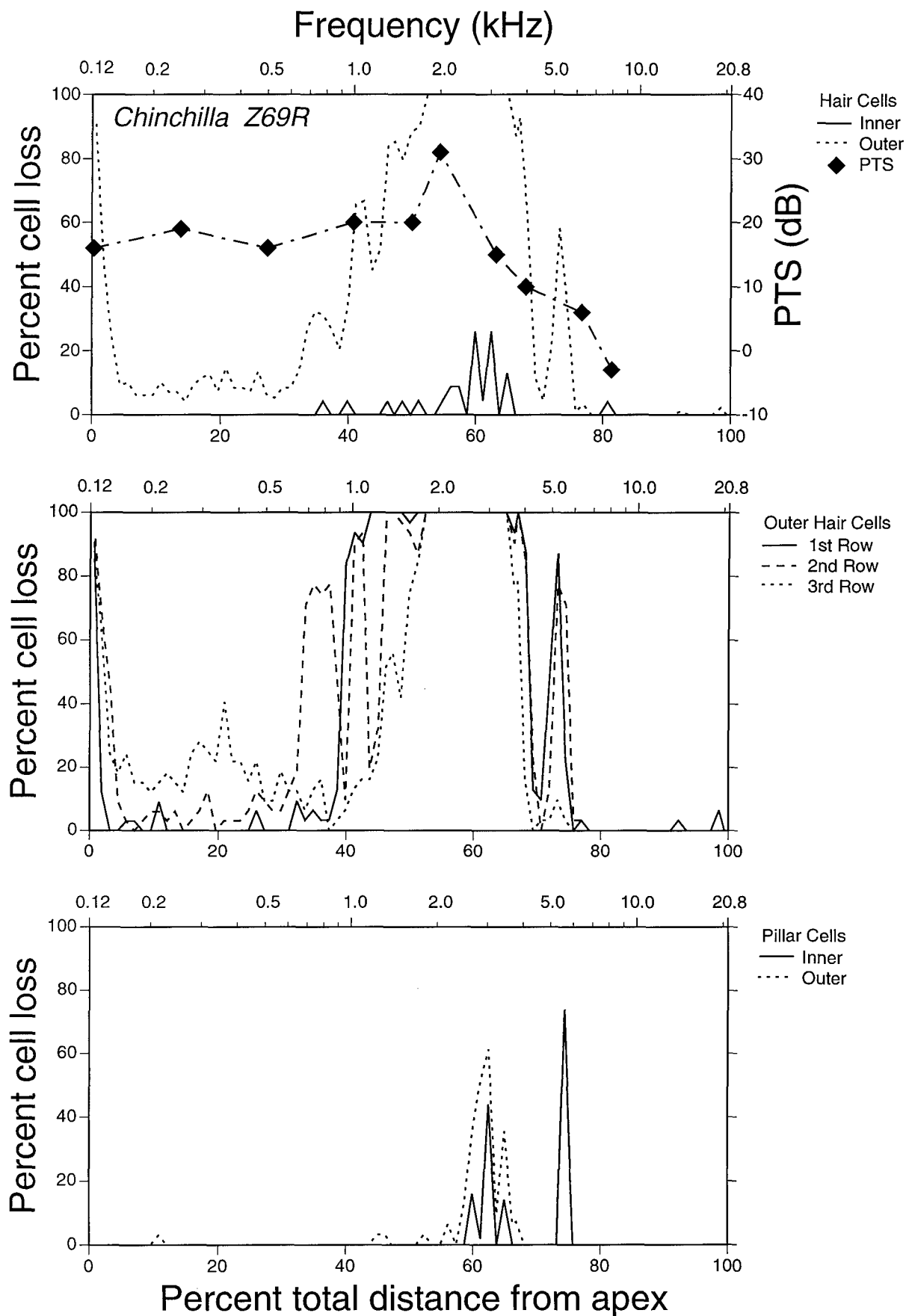


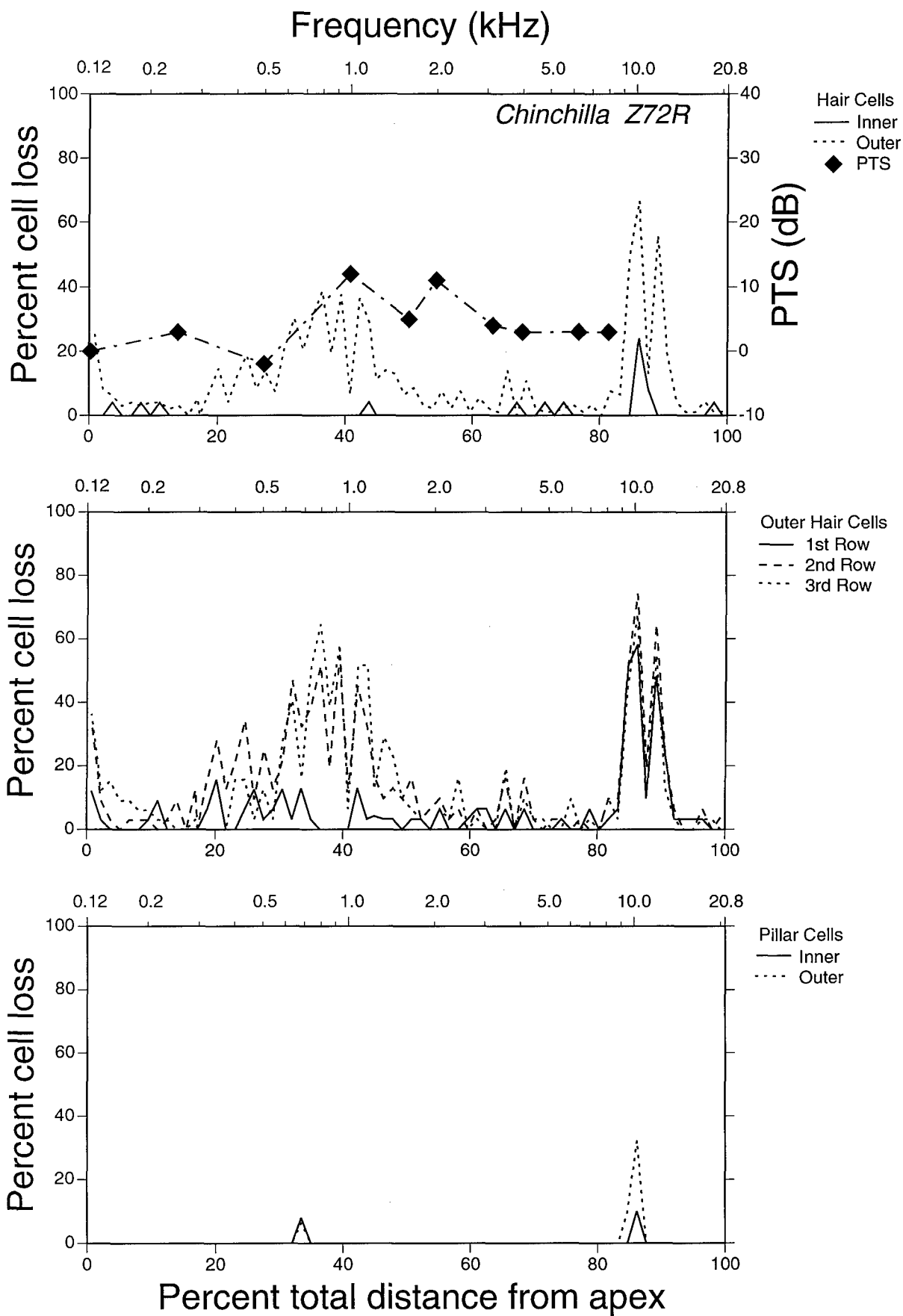


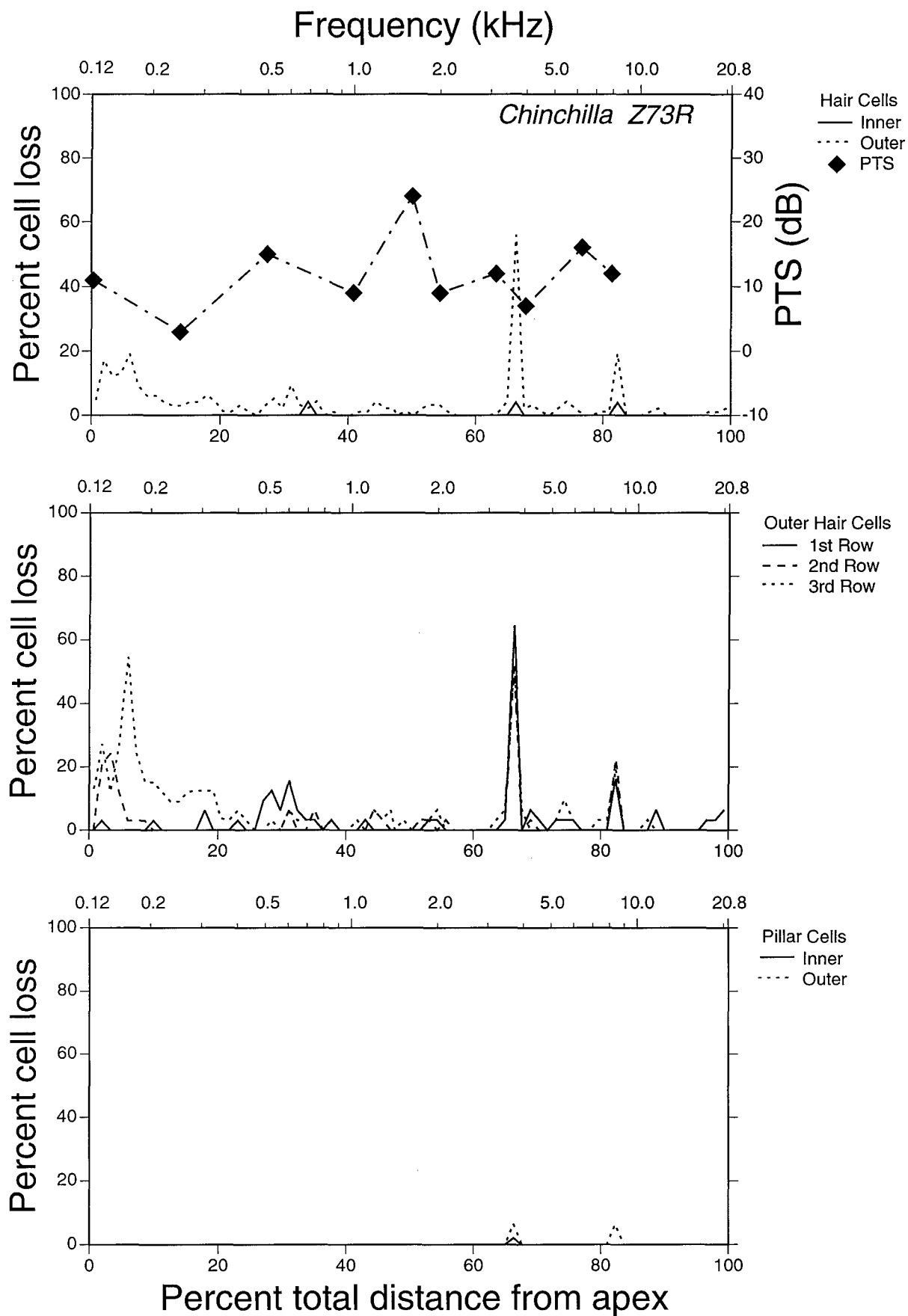




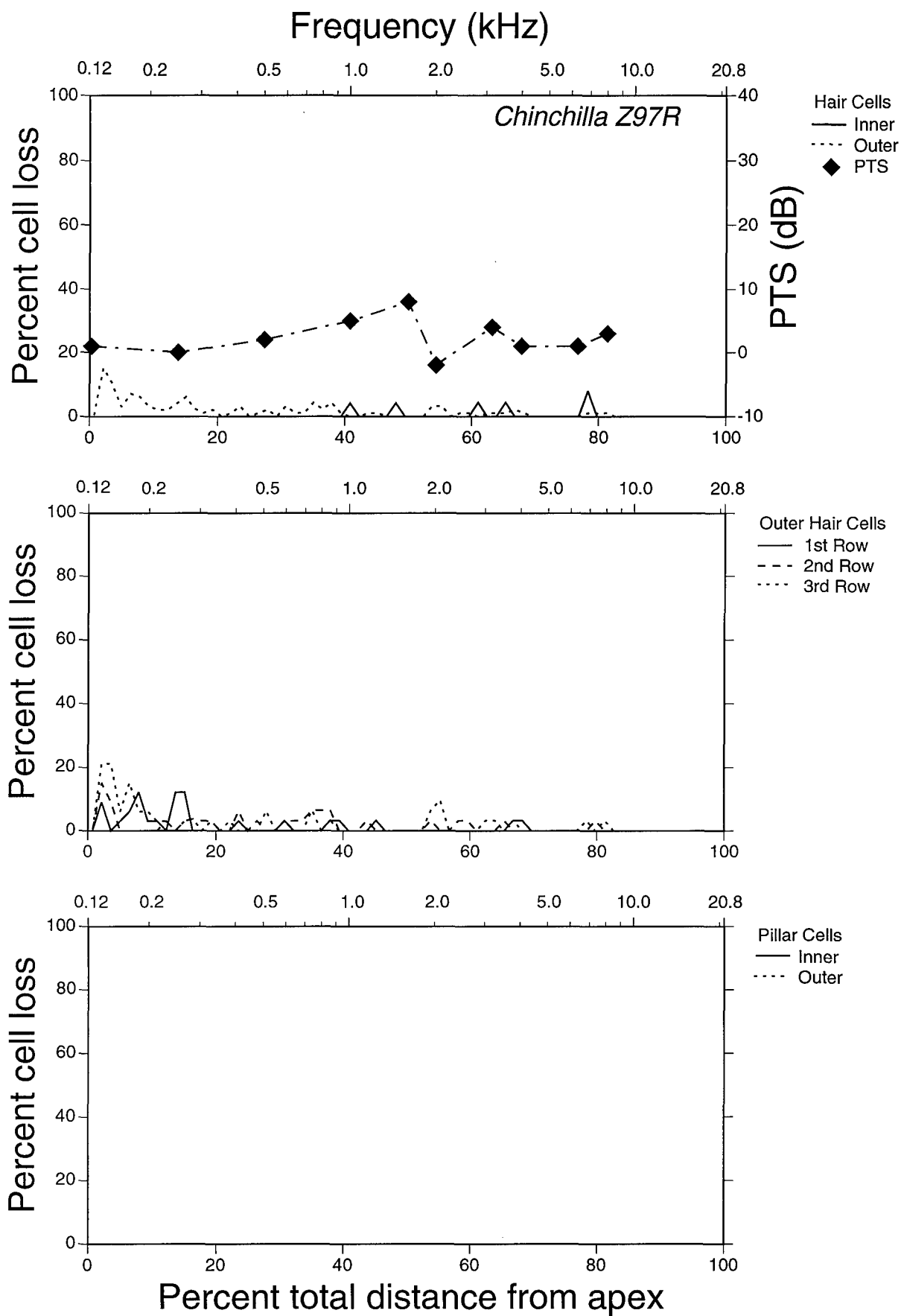






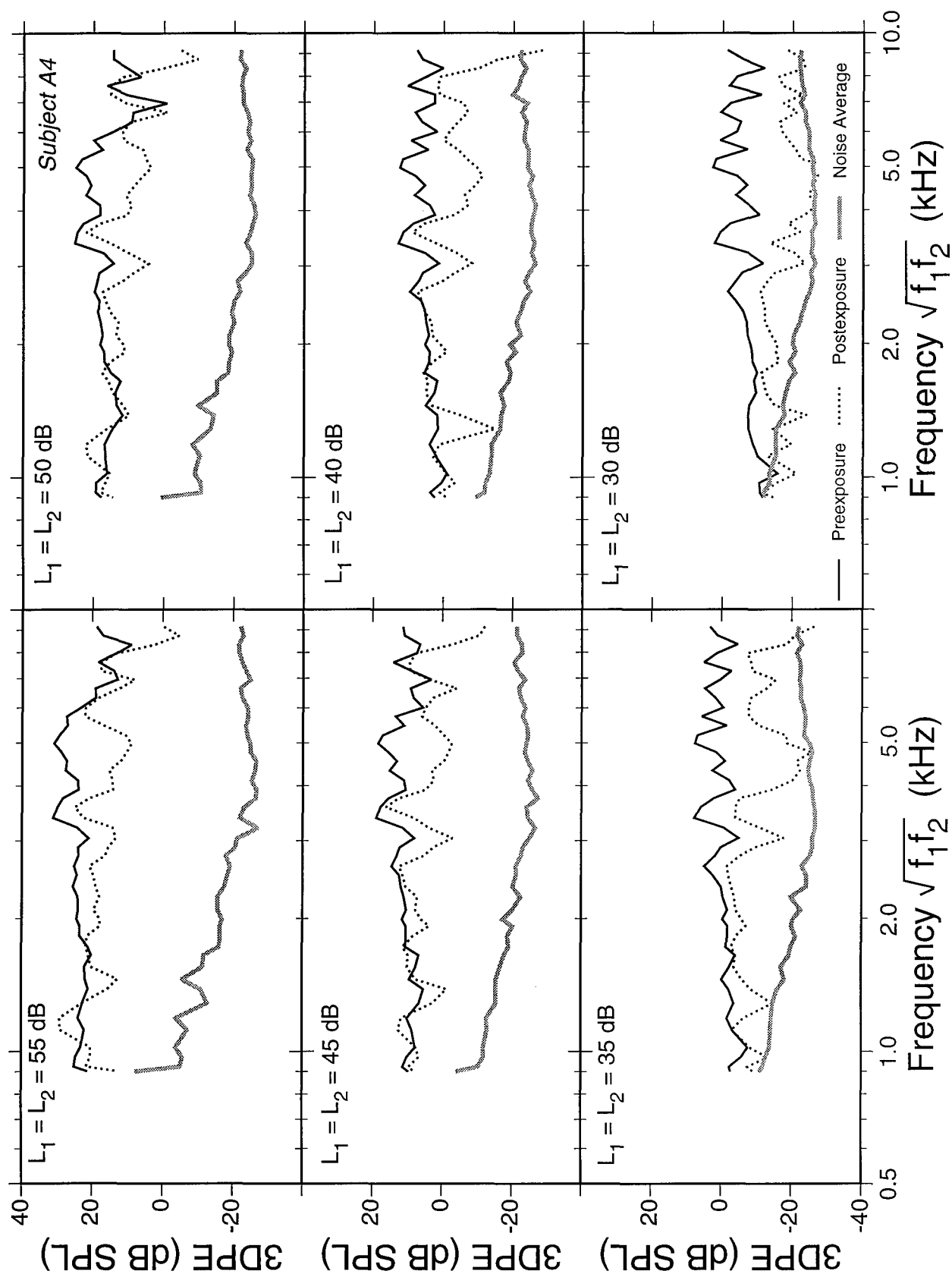


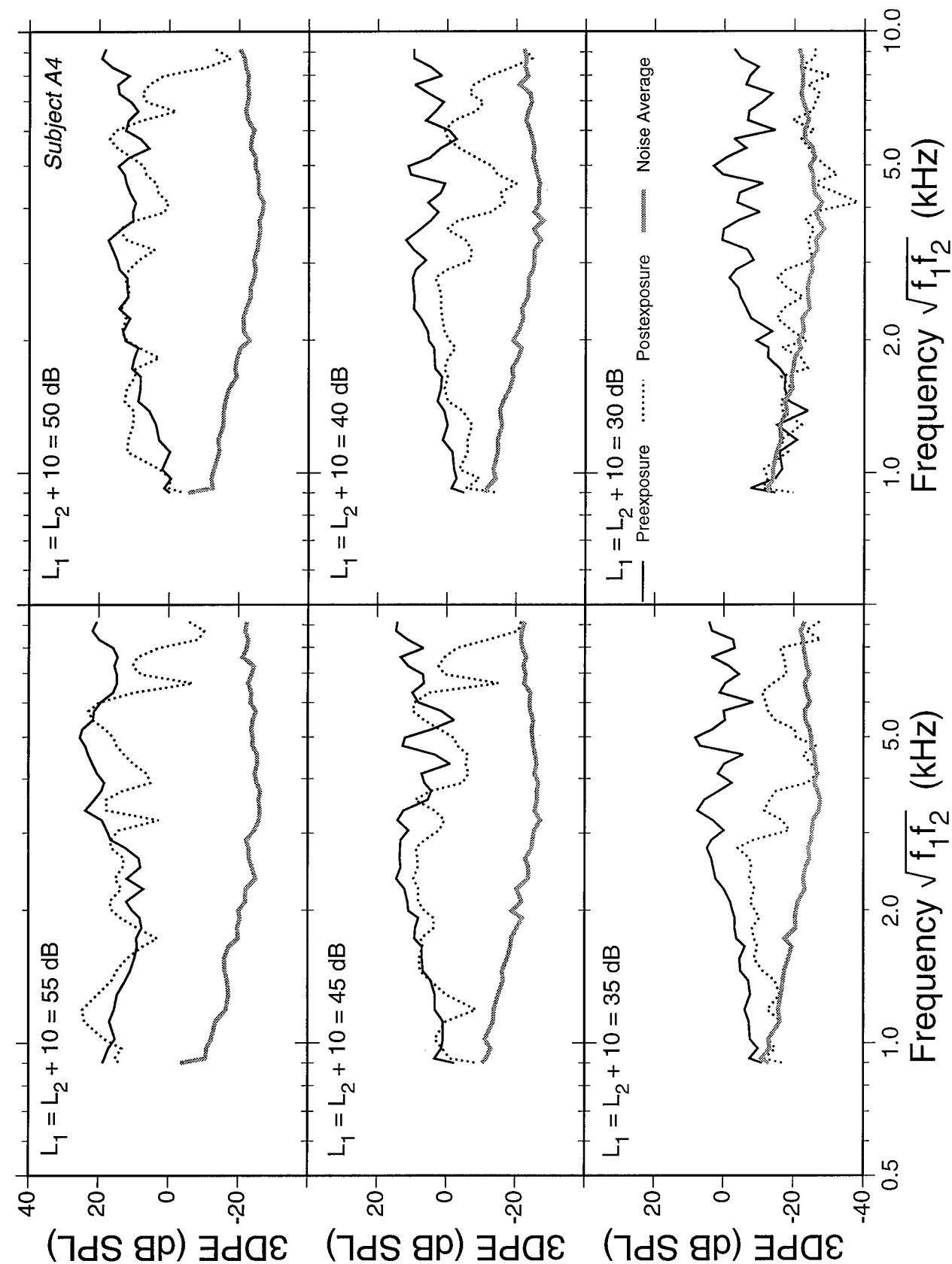


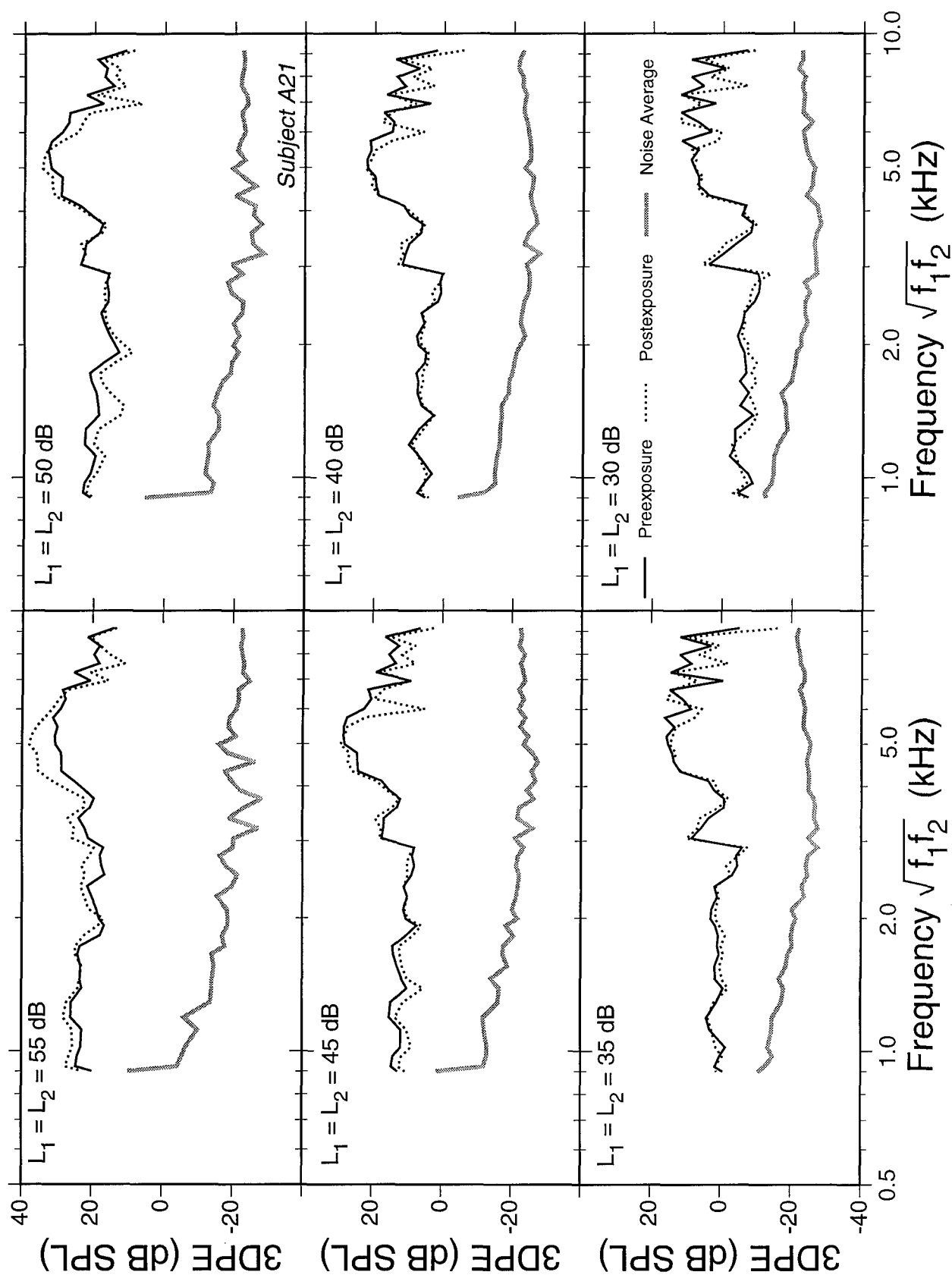


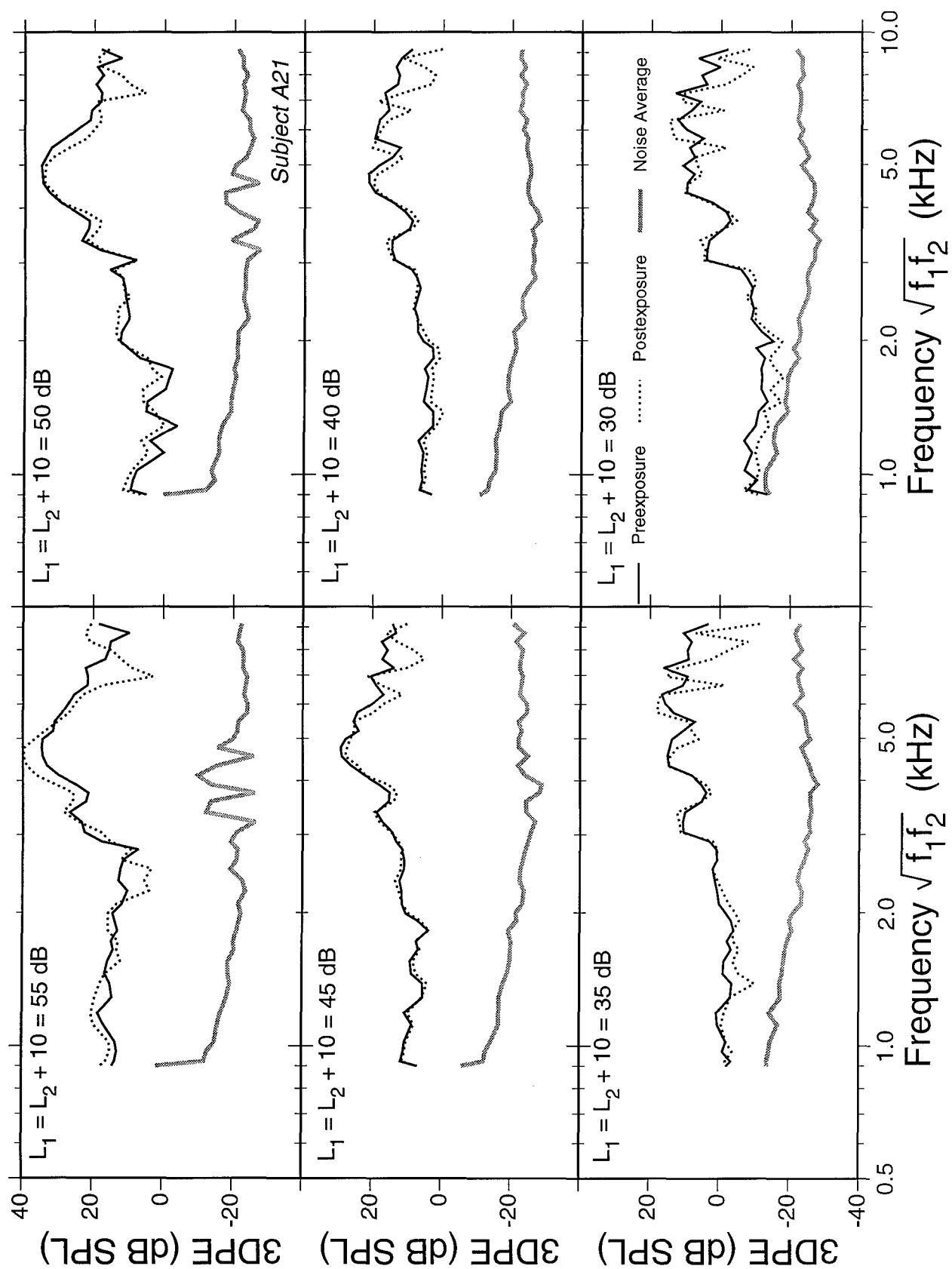
## Individual Subject Pre- and Postexposure DPEgrams

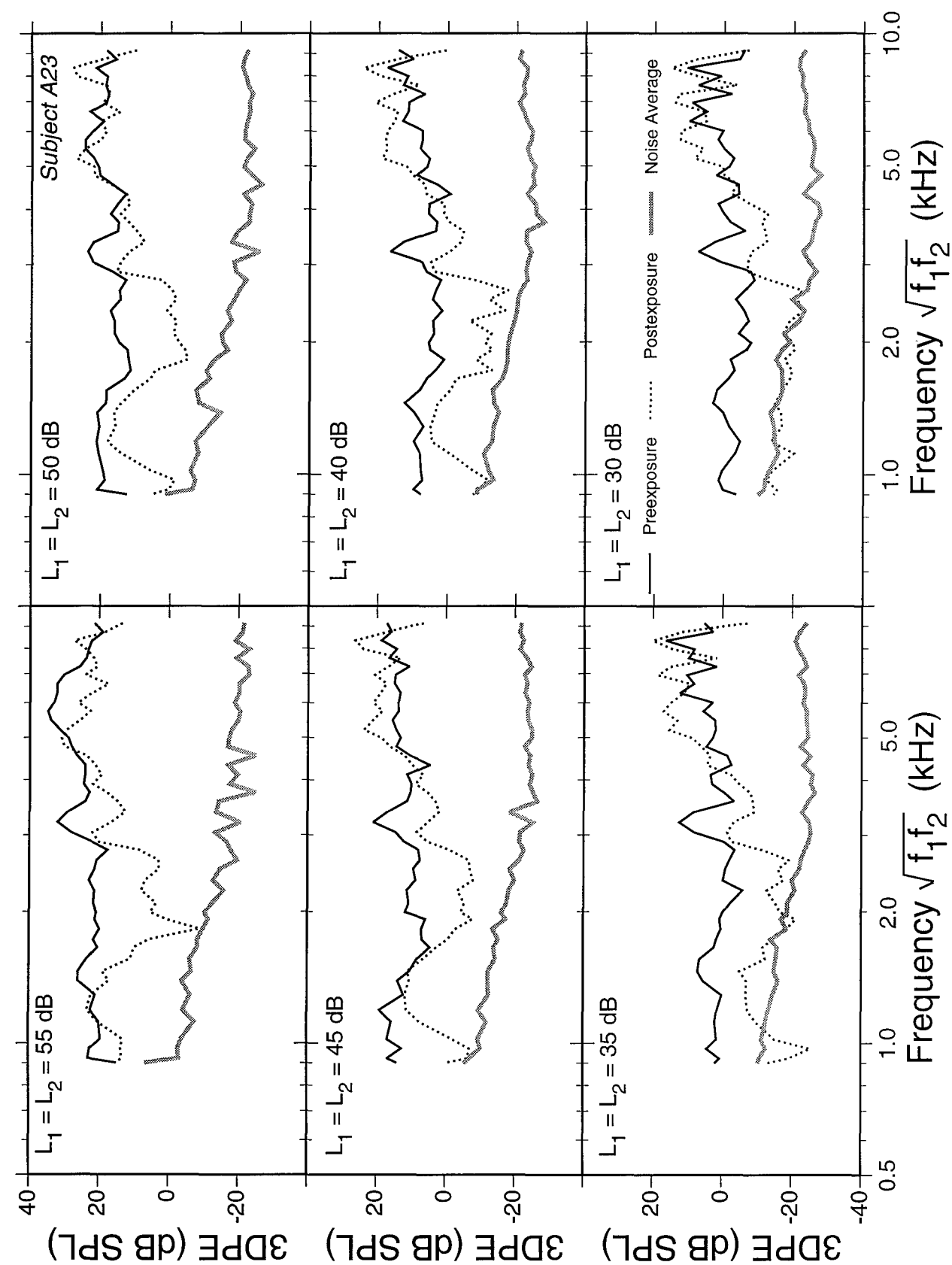
Group mean DPEgrams were presented at the beginning of this data appendix (Pages 72 and 73). The next set of figures (Pages 110 through 129) show the pre- and postexposure DPEgrams for the 10 individual animals that make up this group (see Page 68). The solid lines represent the mean preexposure DPEgram at the six equal-primary measurements ( $L_1 = L_2 = 55$  to 30 dB SPL) and six unequal-primary ( $L_1 = L_2 + 10 = 55$  to 30 dB SPL) measurements. Each subject's DPEgrams represent the average of three measurements made on different days. The dashed lines represent the group mean postexposure measurements made at least 30 days following exposure. The thick gray line represents the average noise floor over the pre- and postexposure measurements.

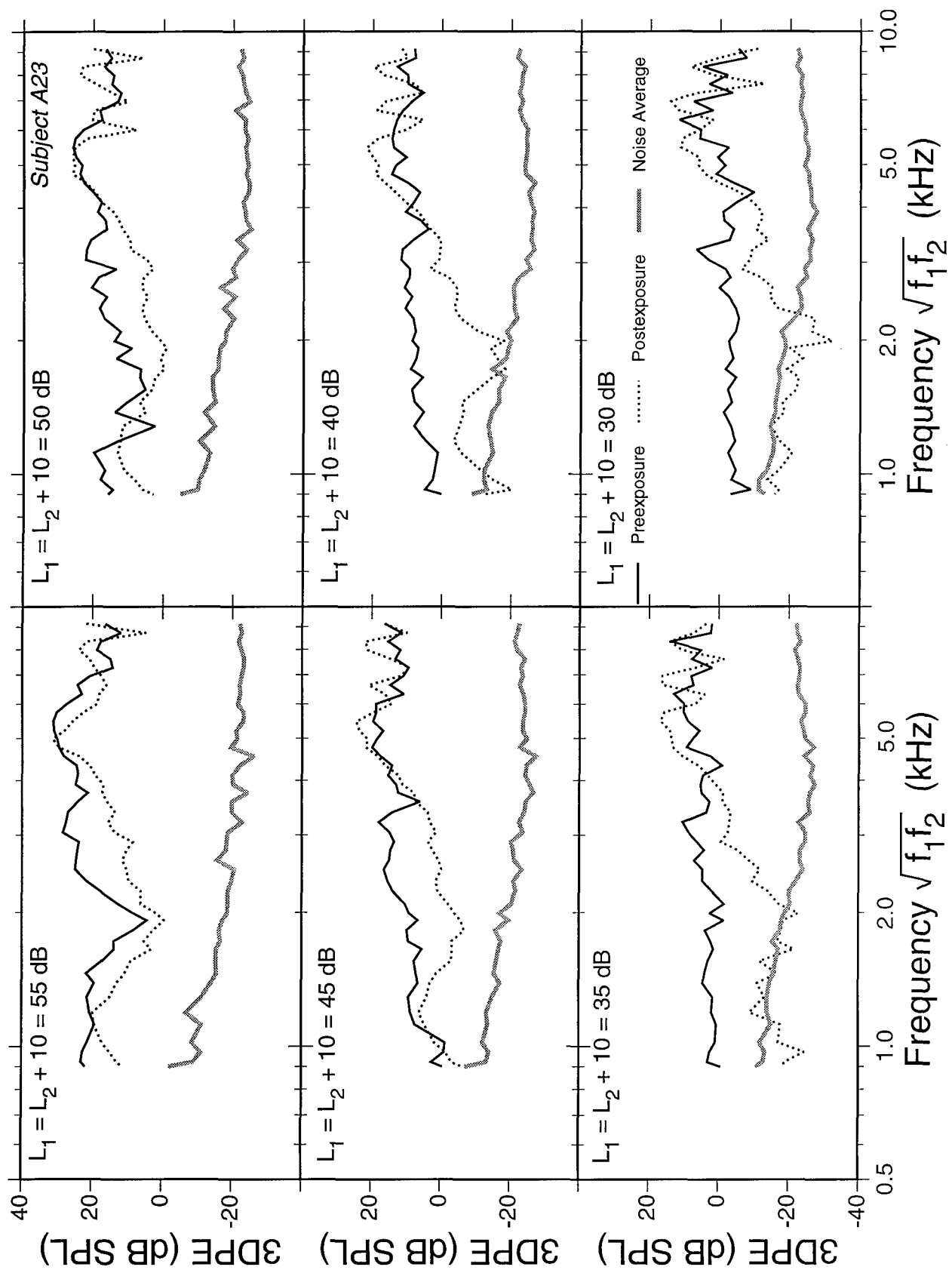




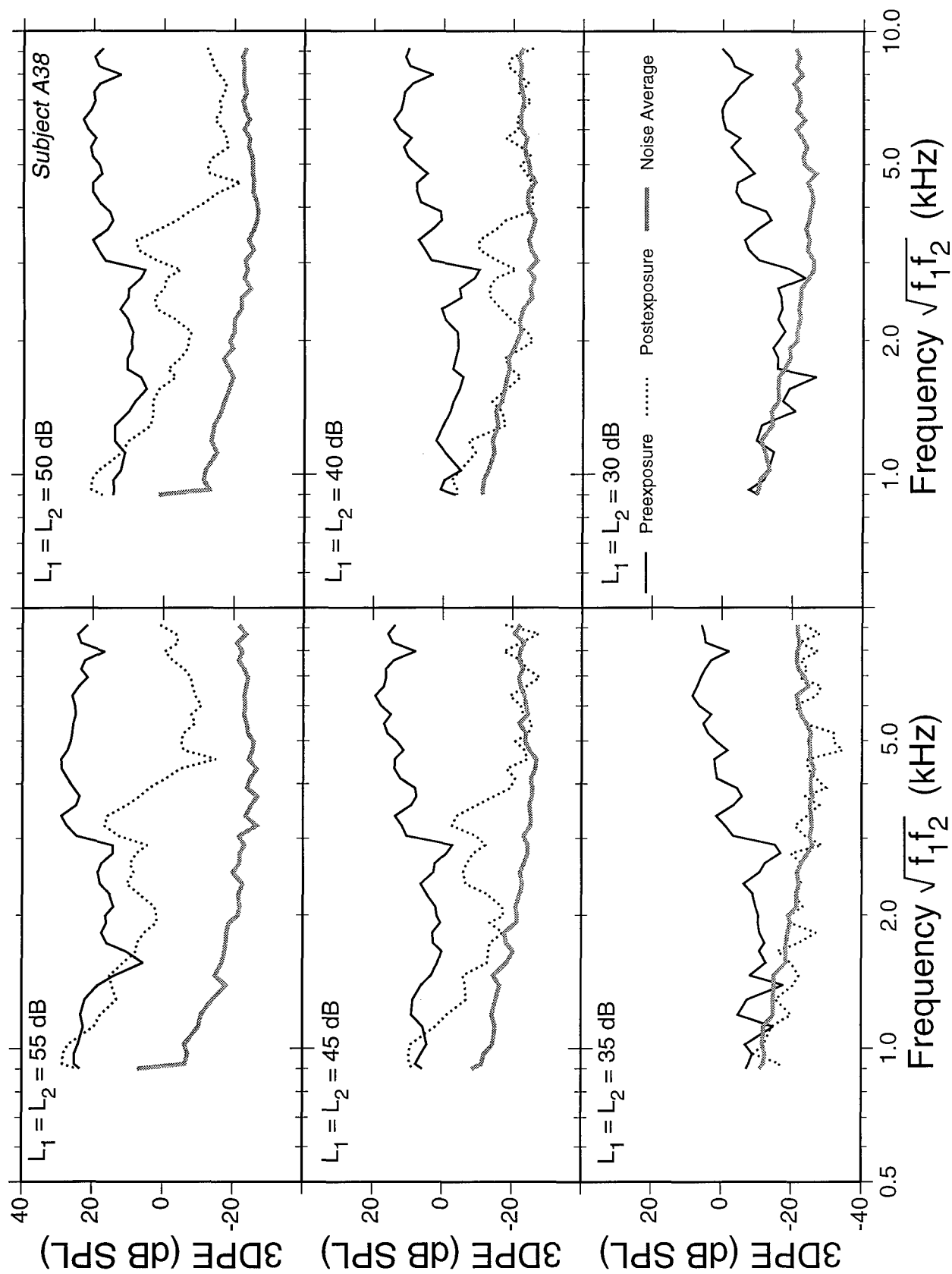


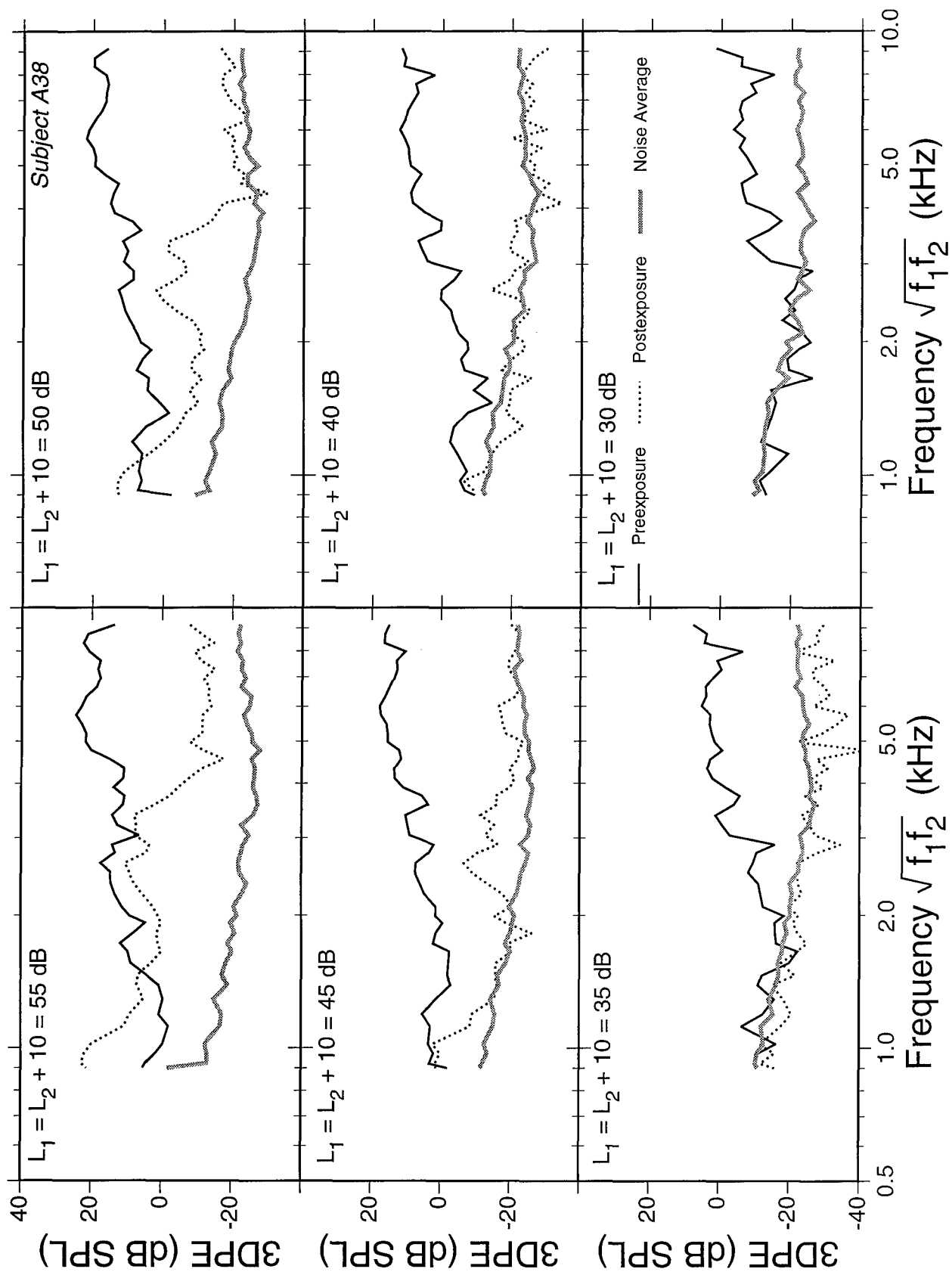


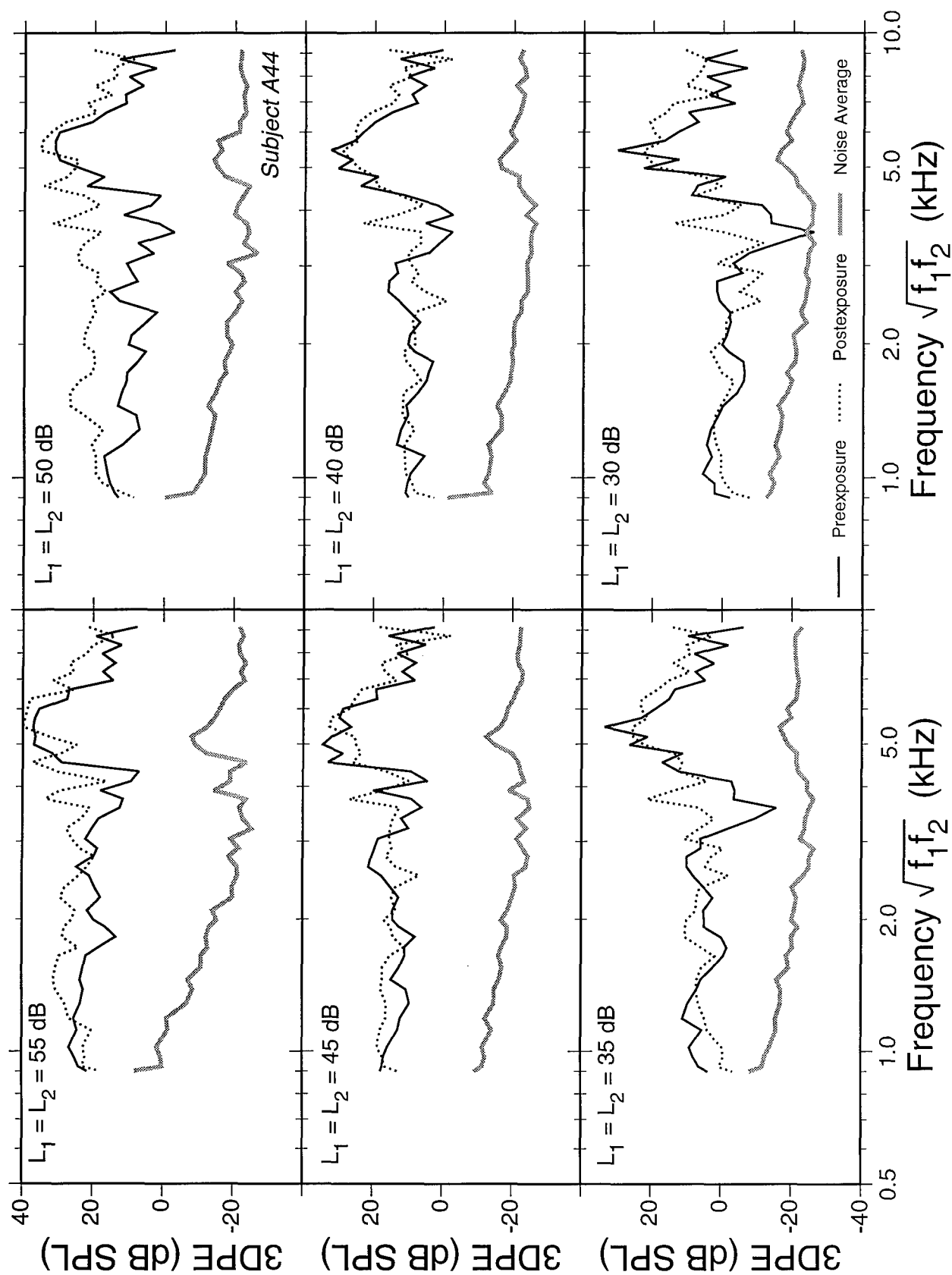


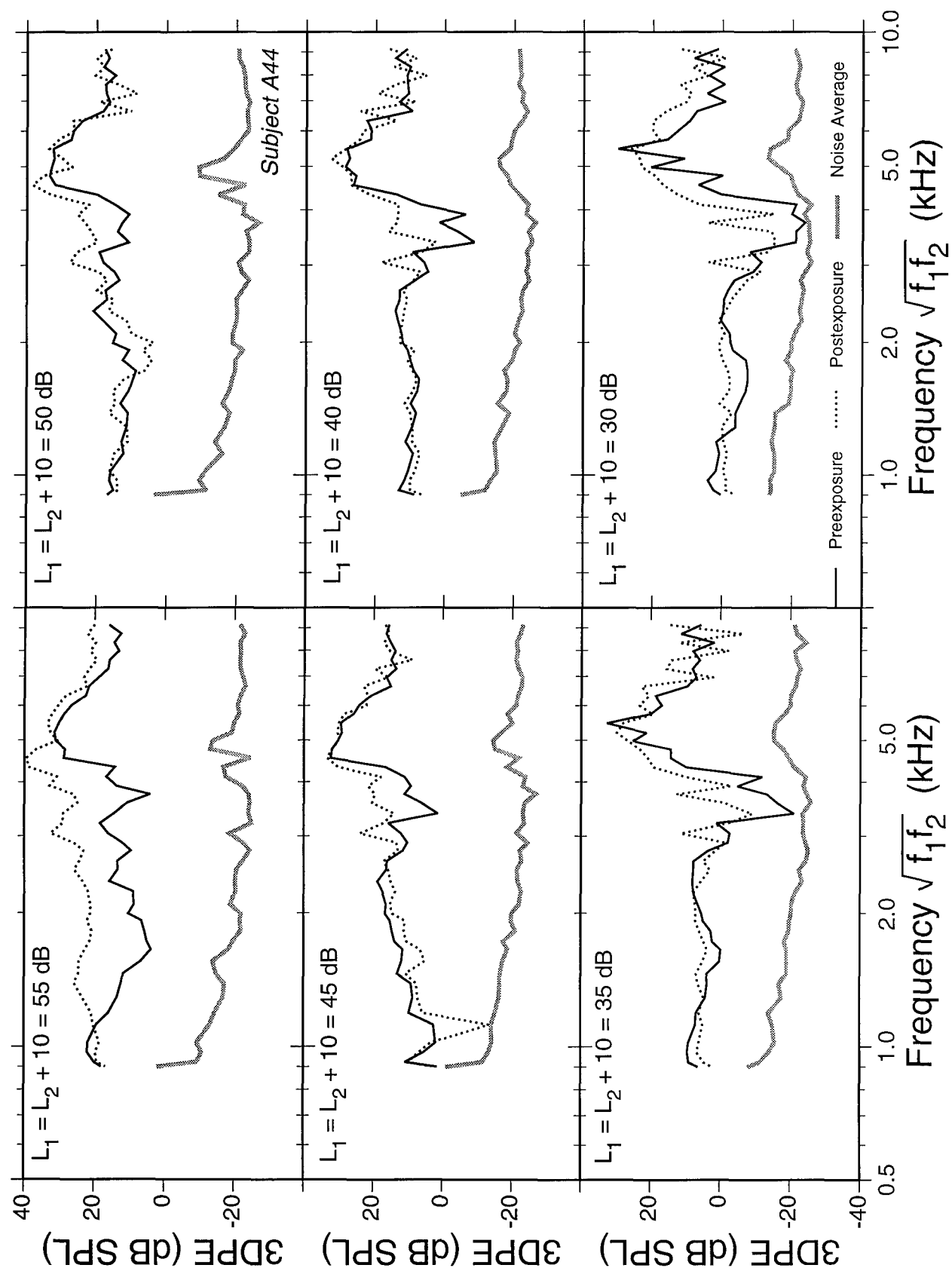


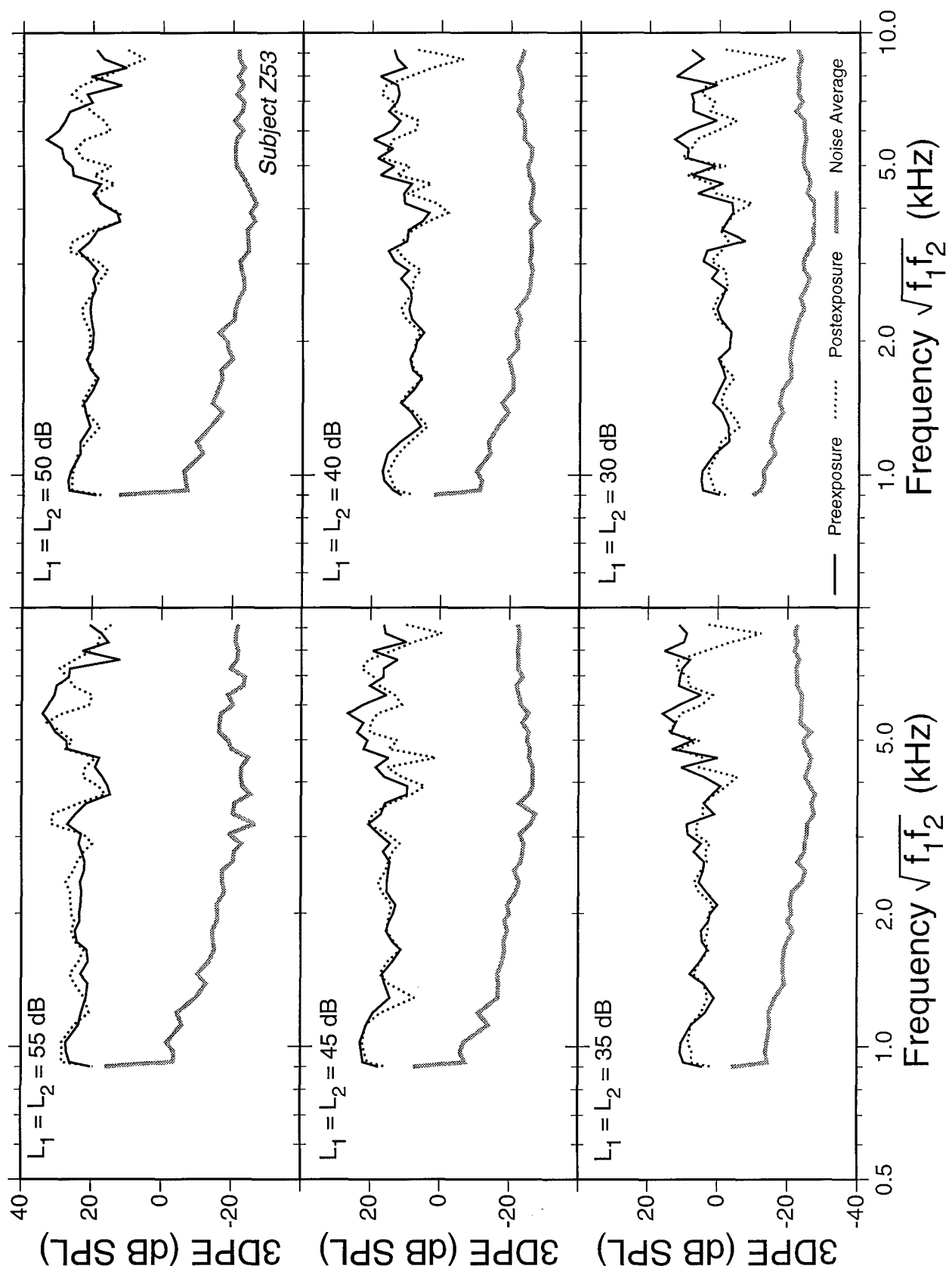


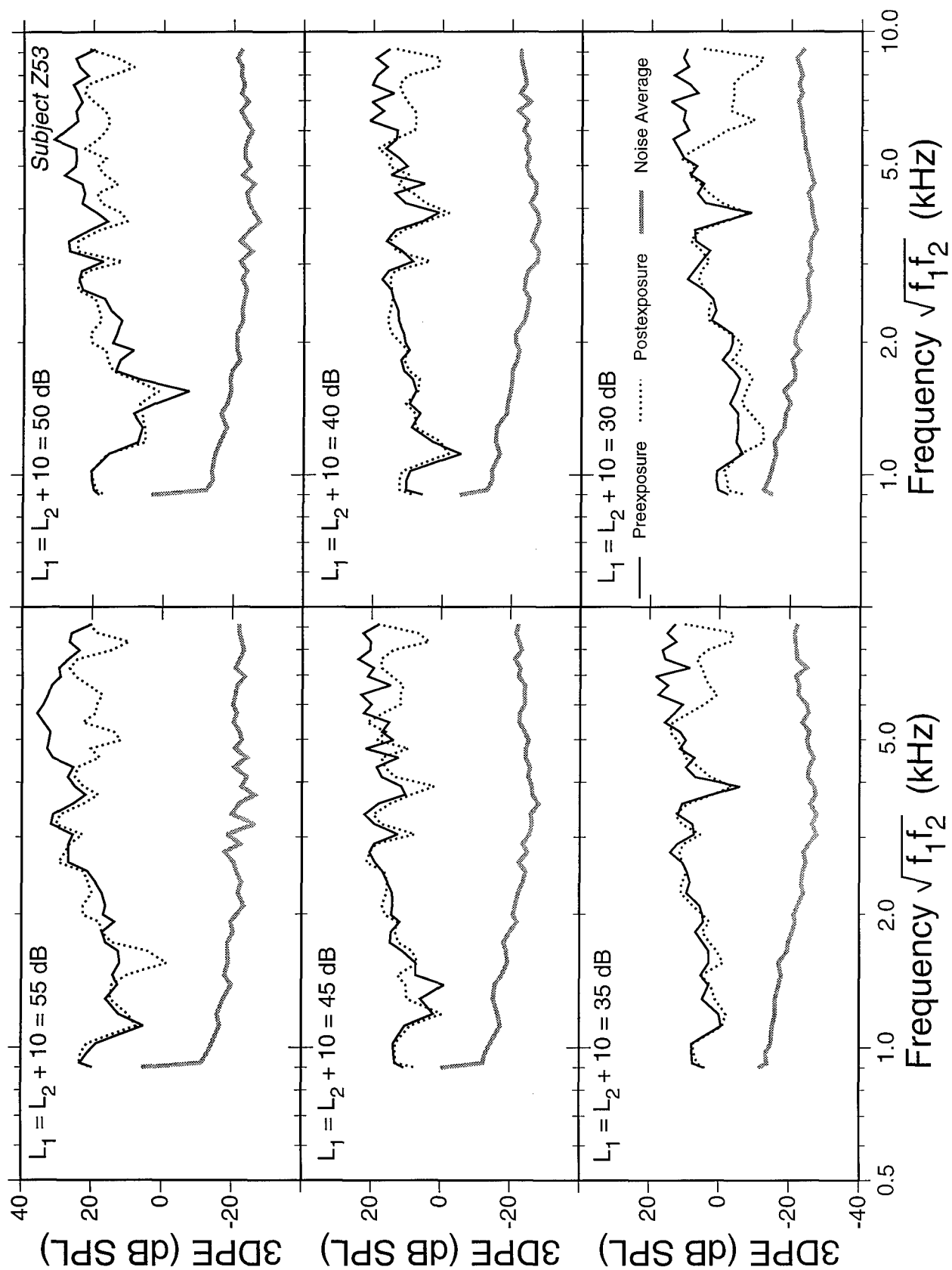


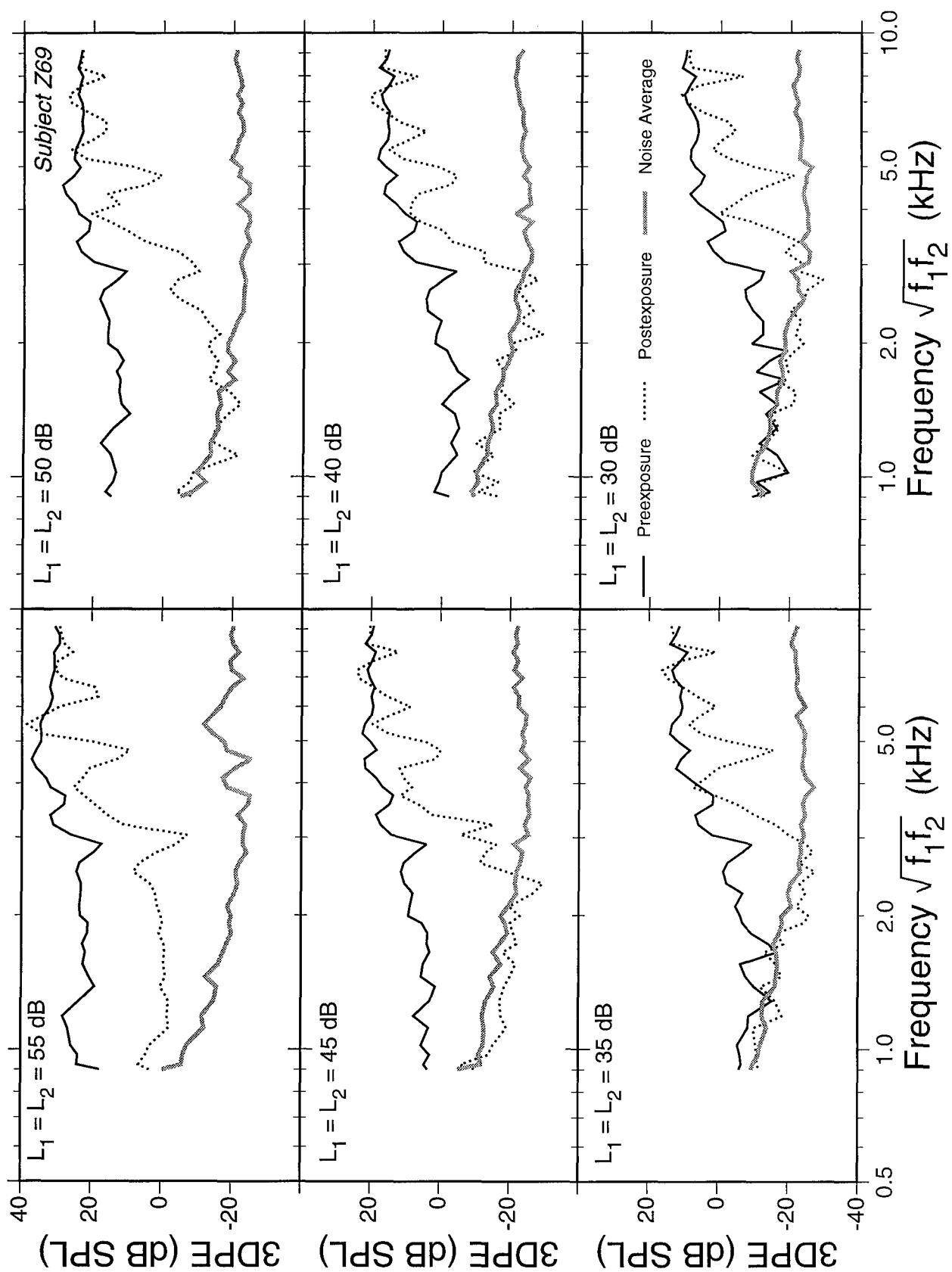


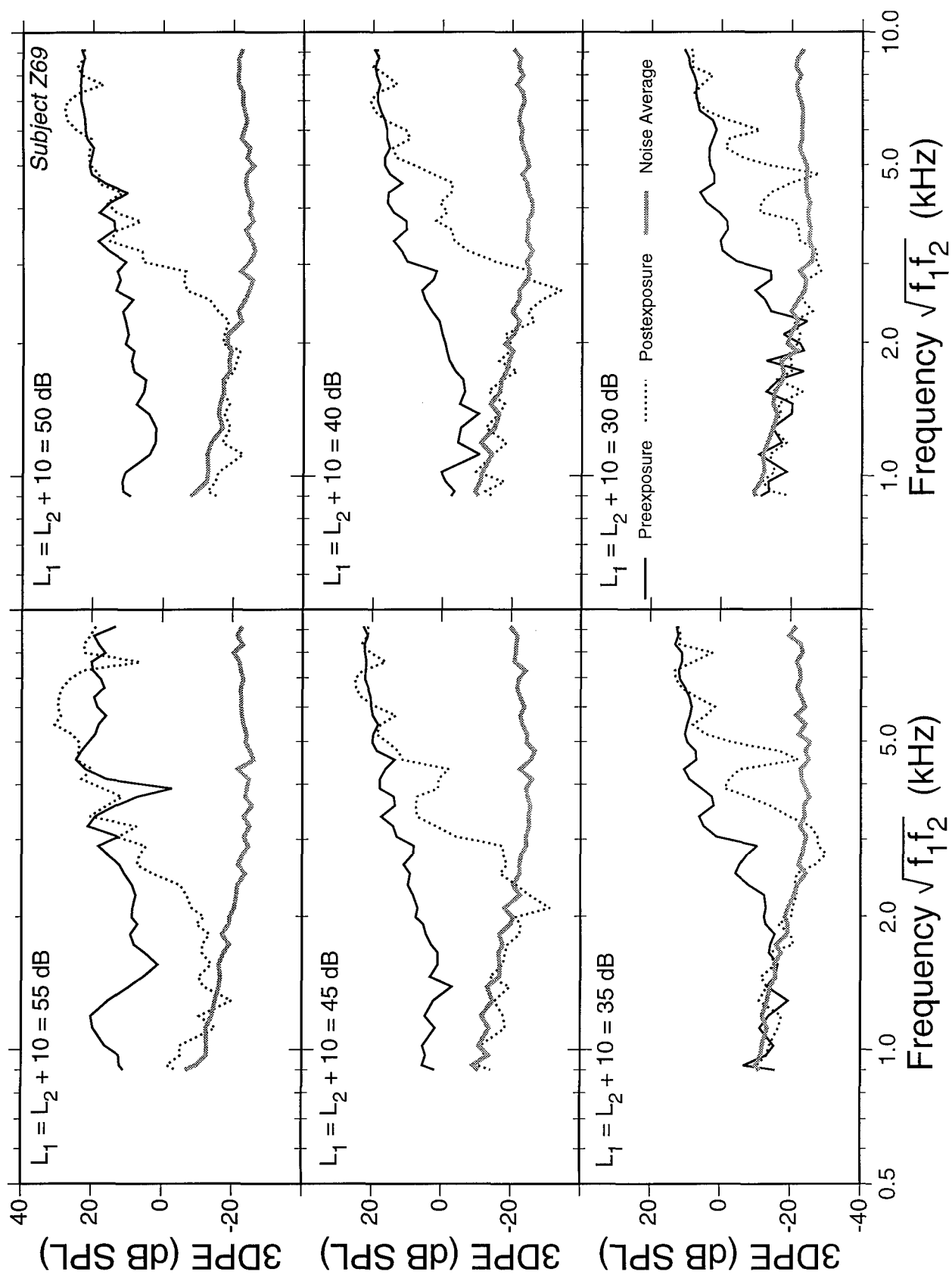




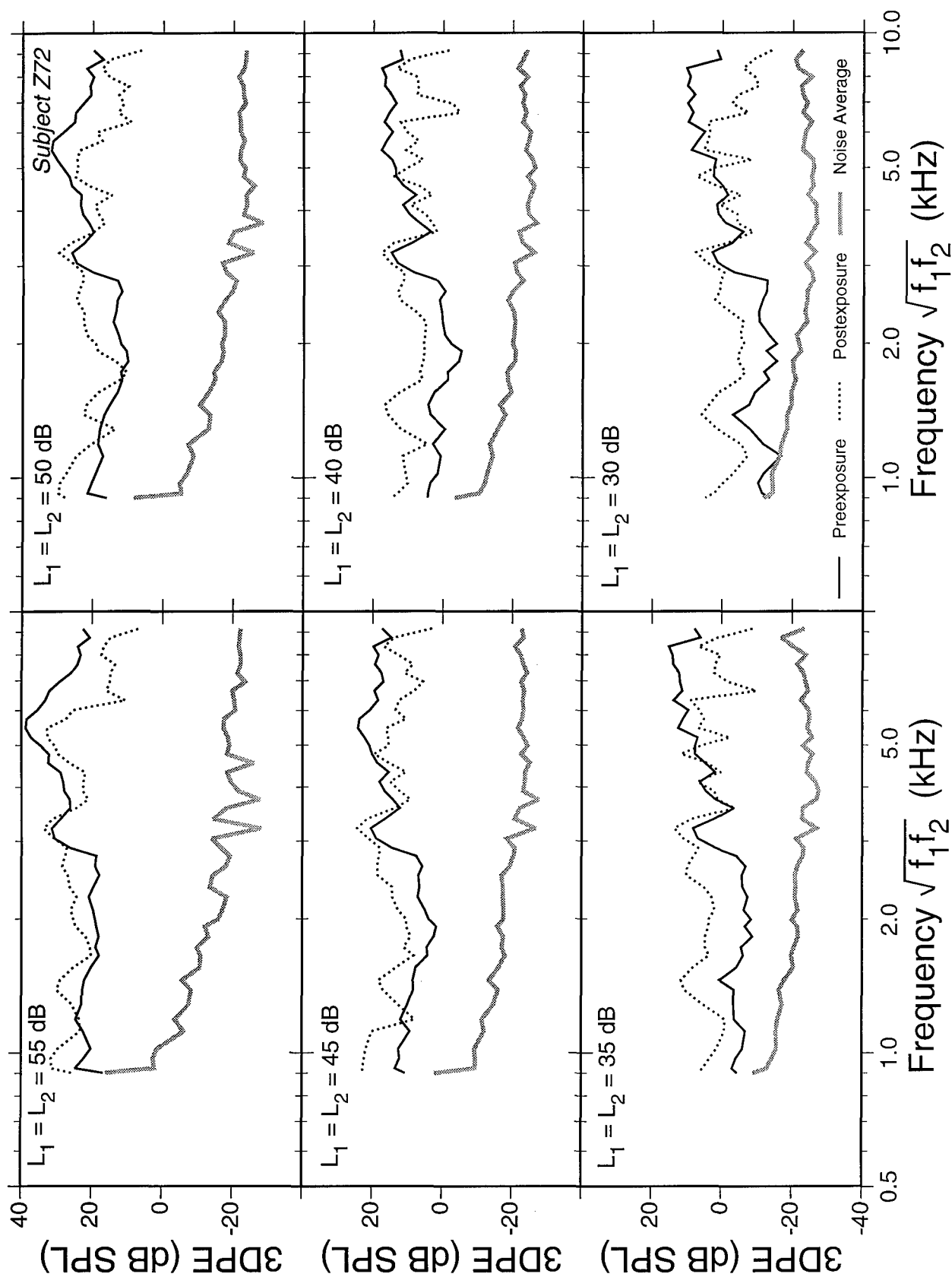


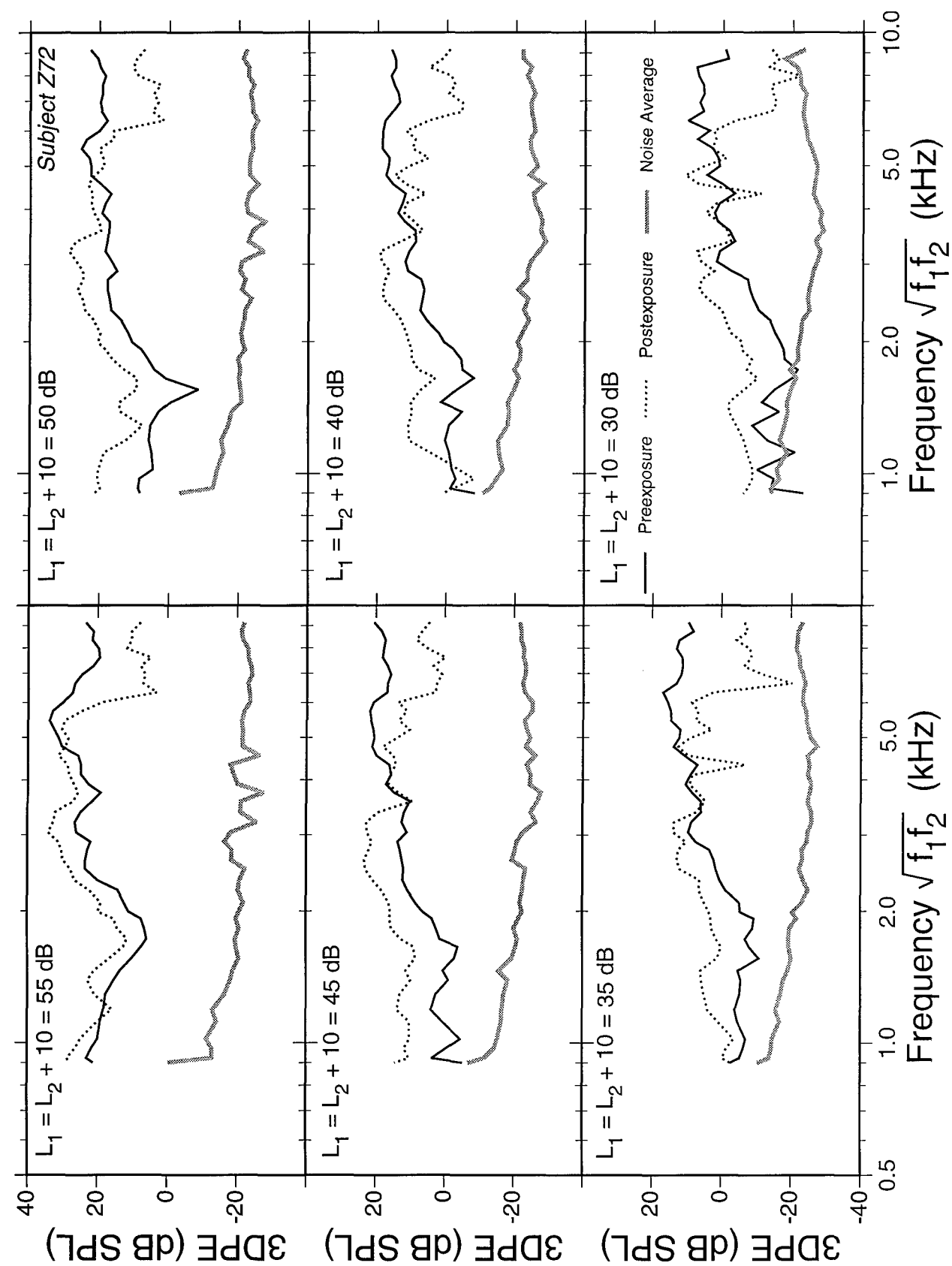


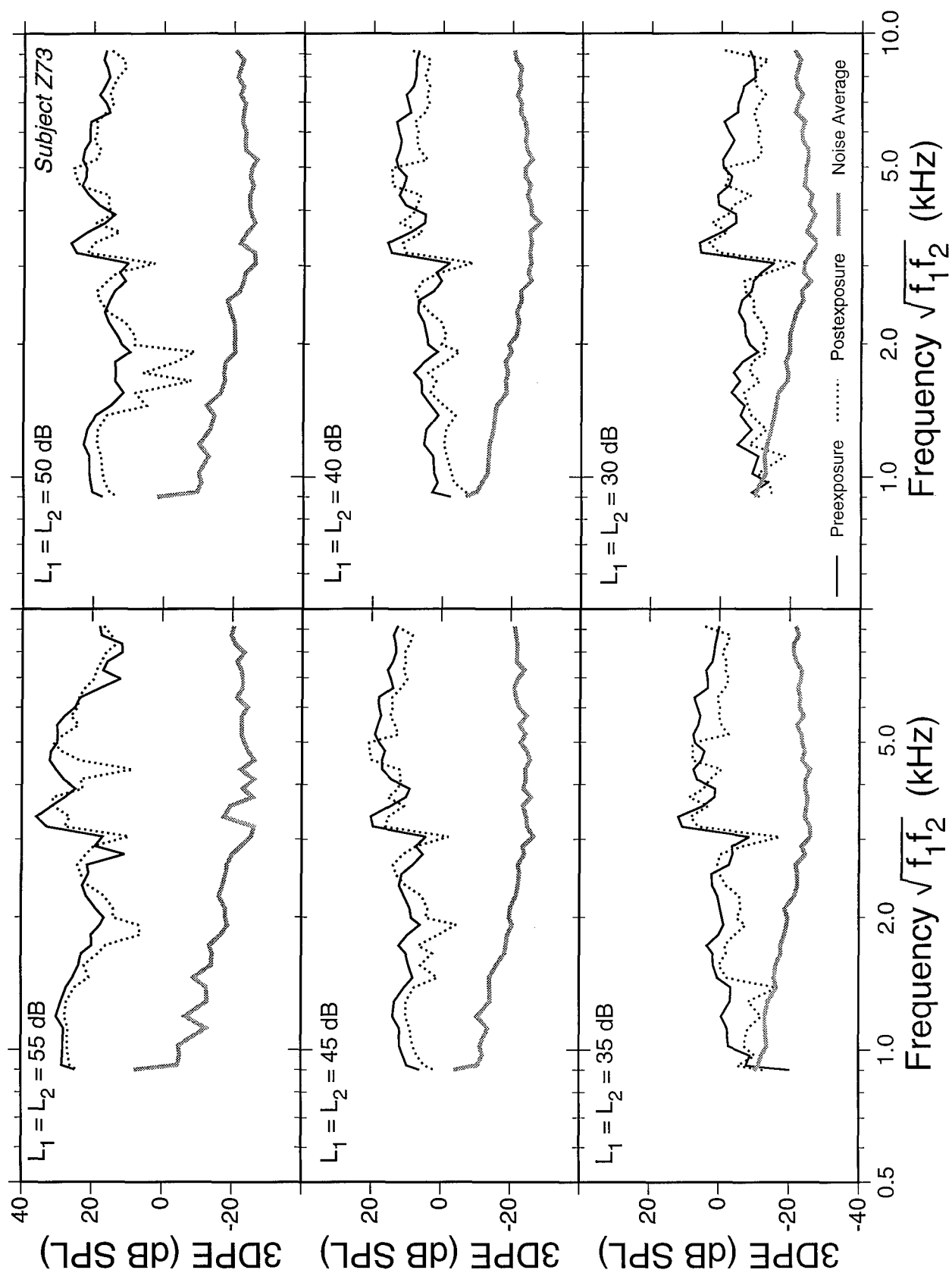


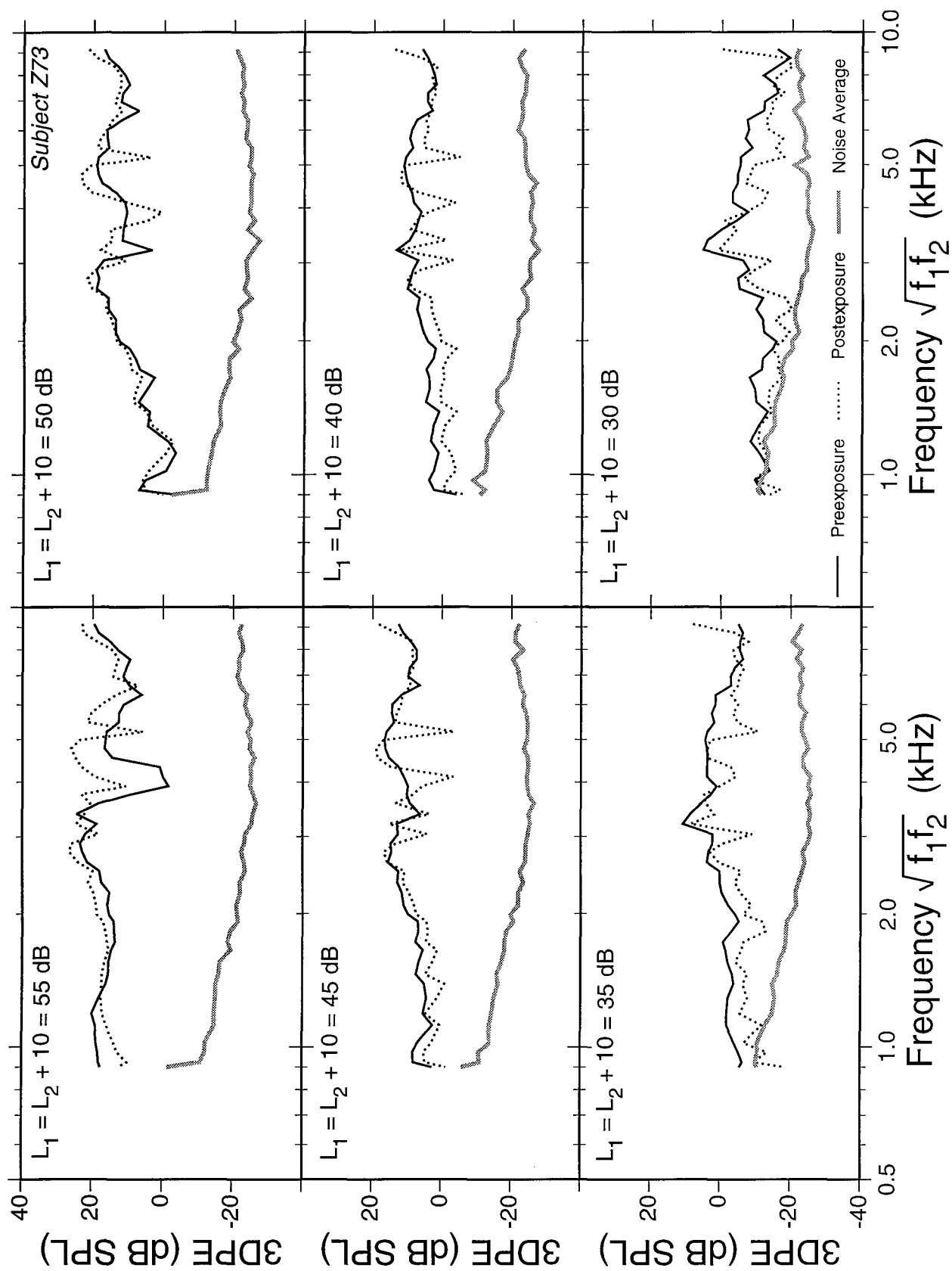


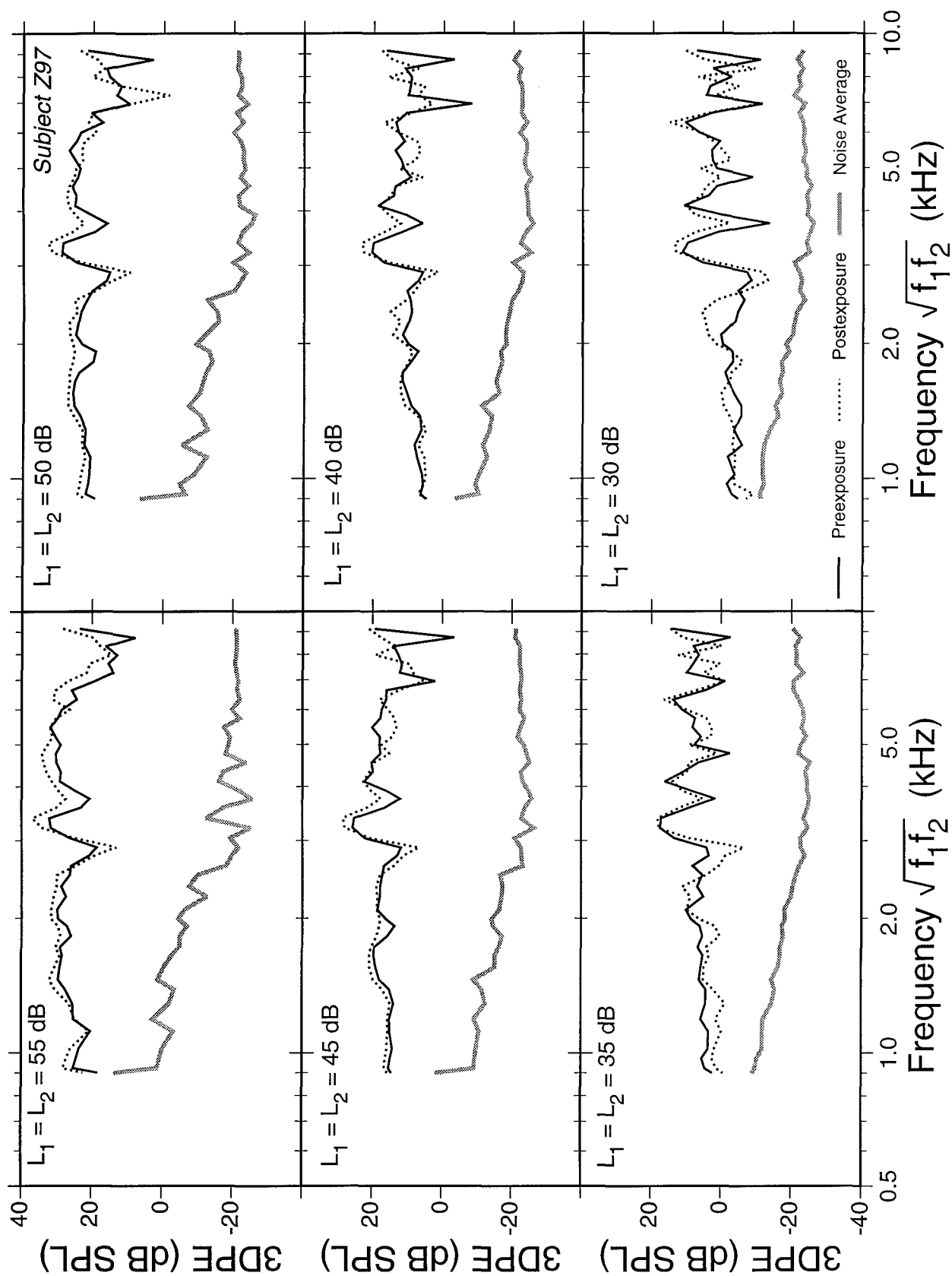


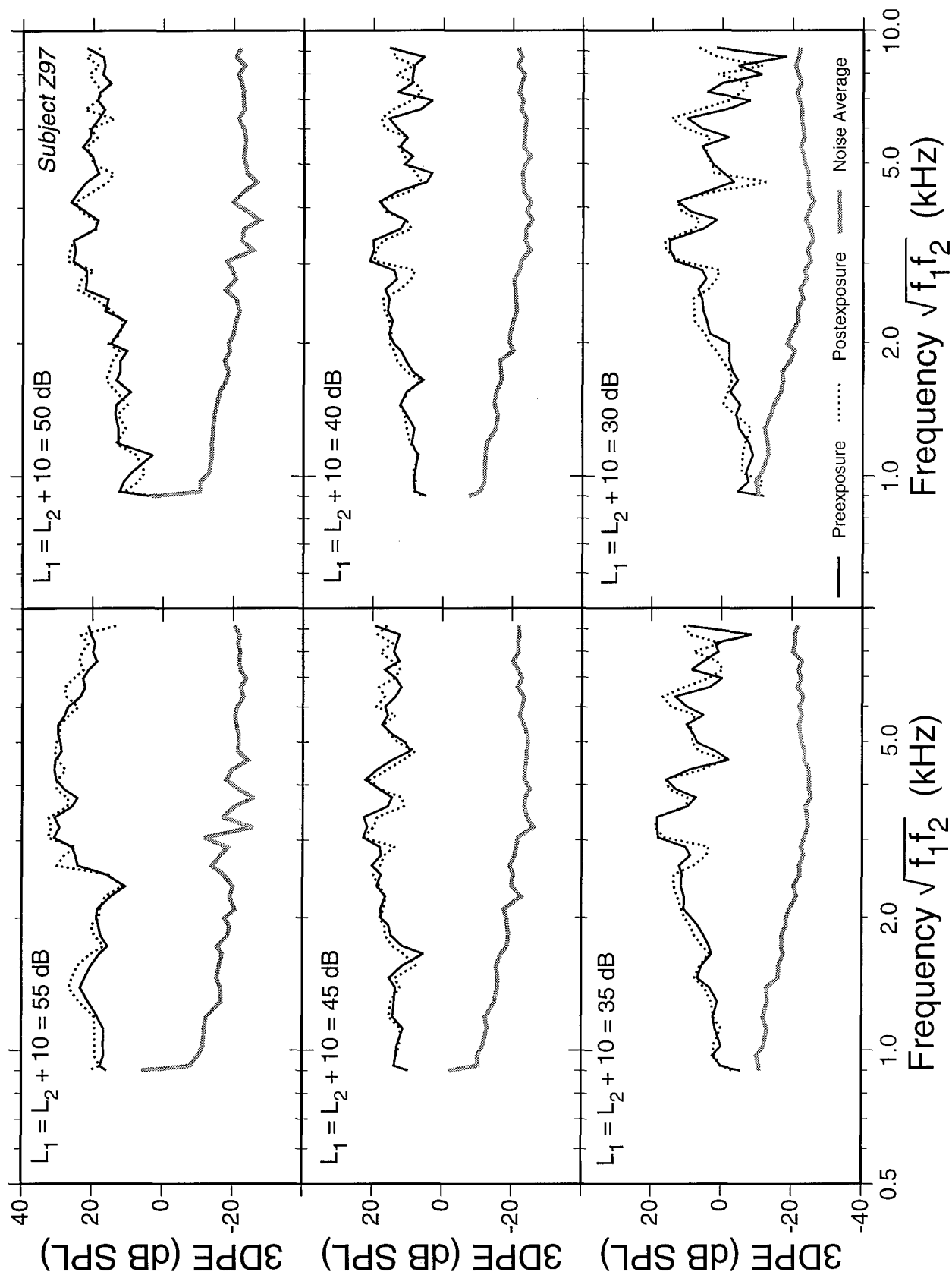






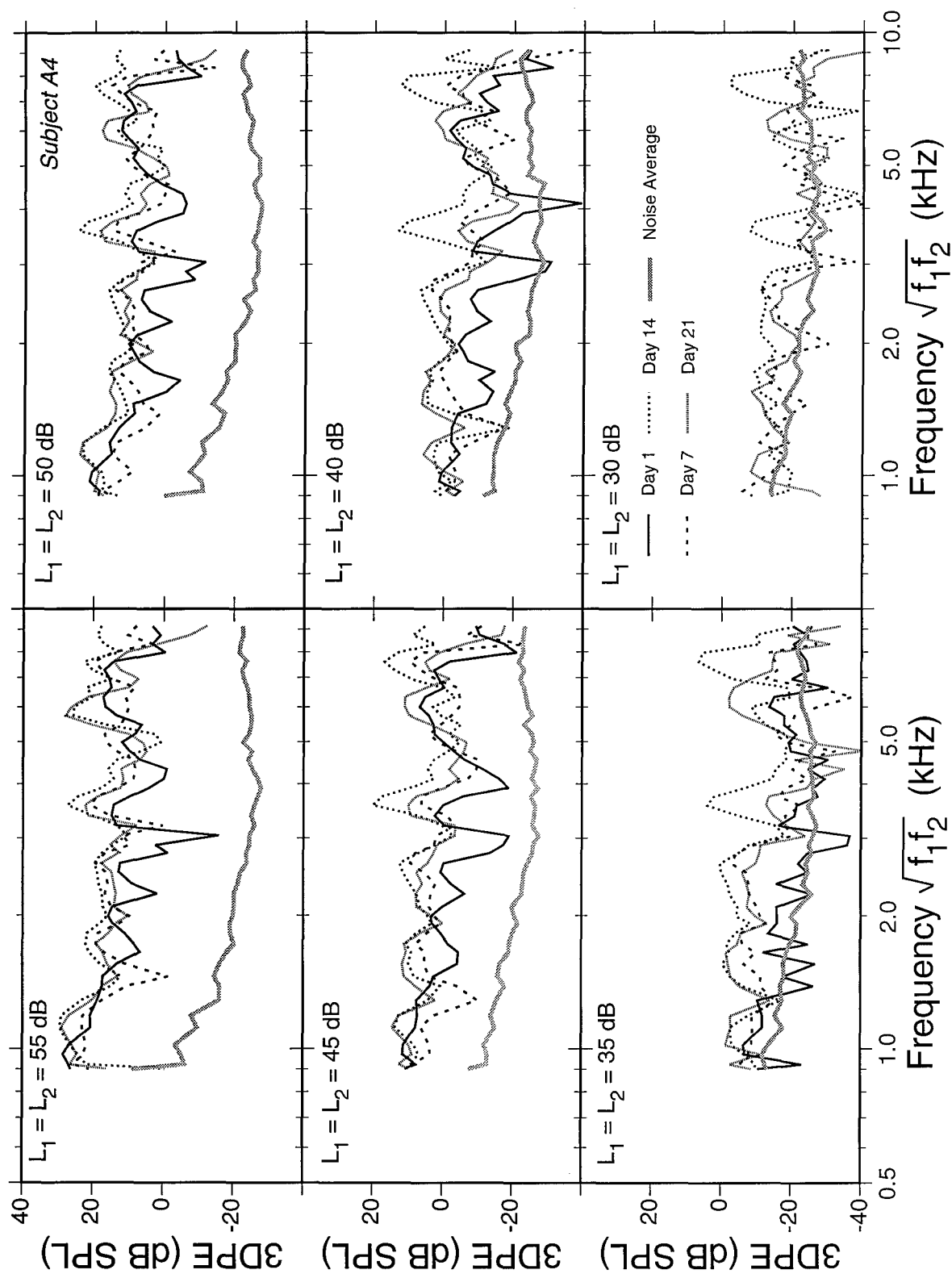




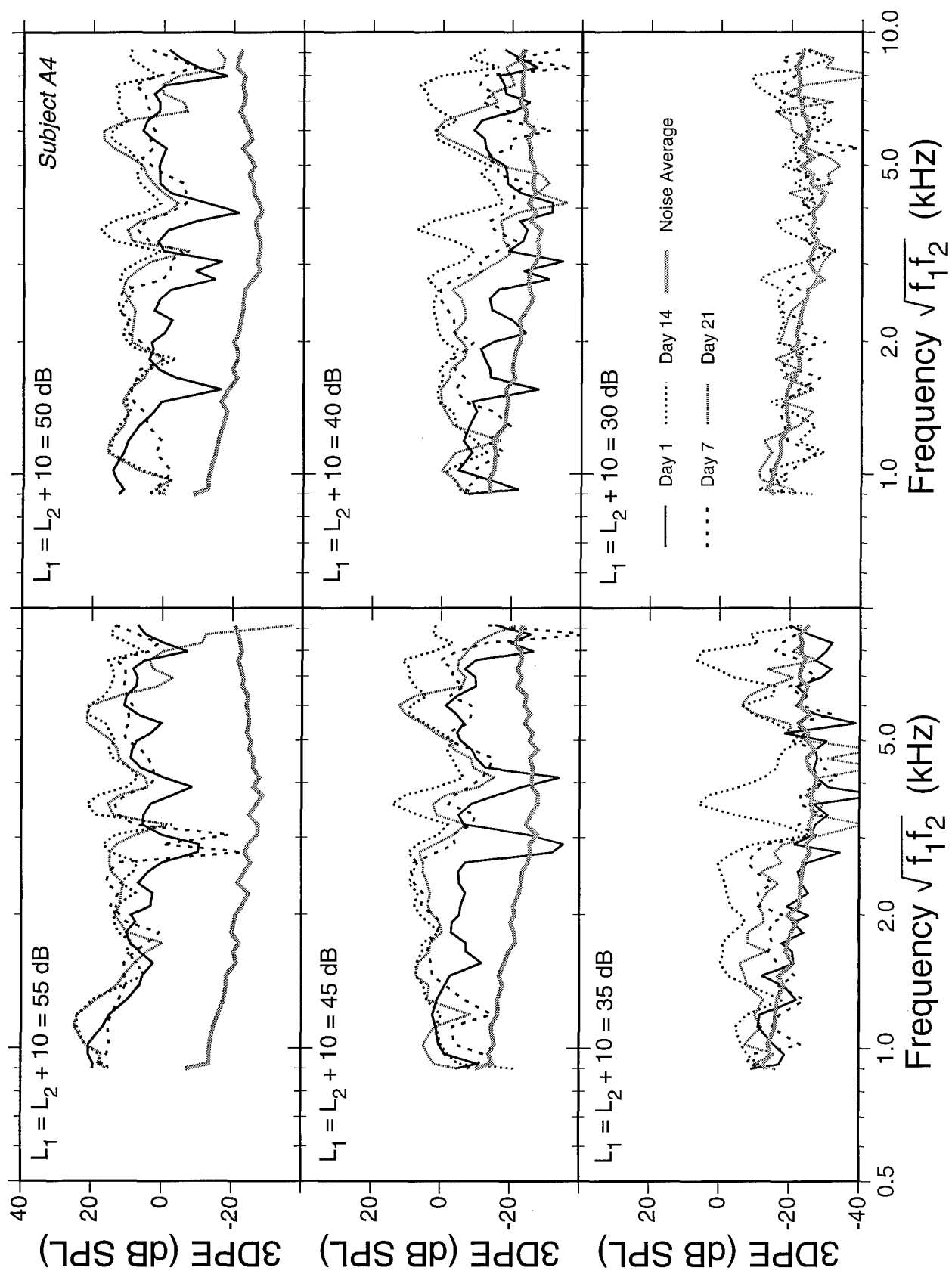


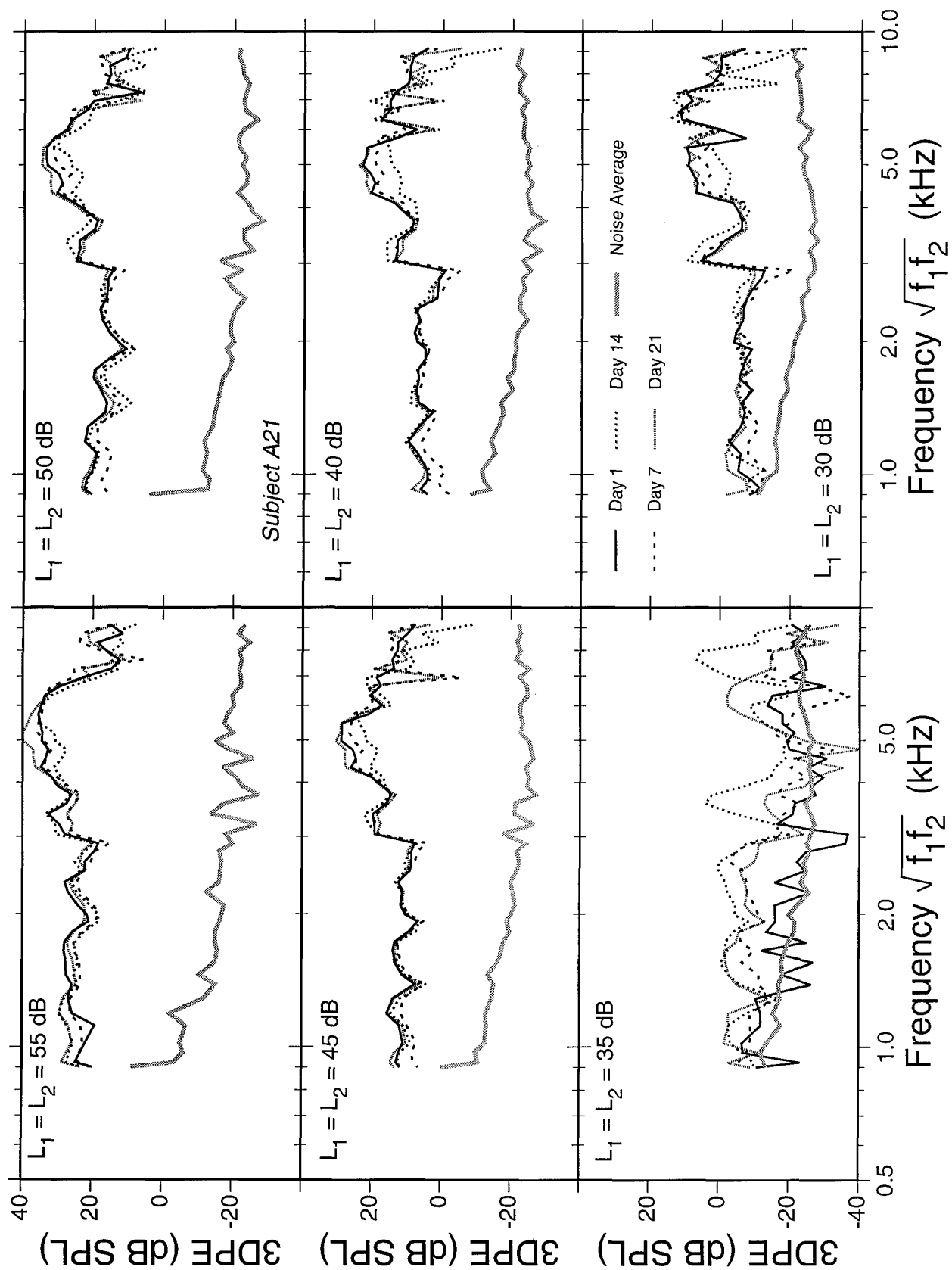
## Individual Subject DPEgrams During Recovery

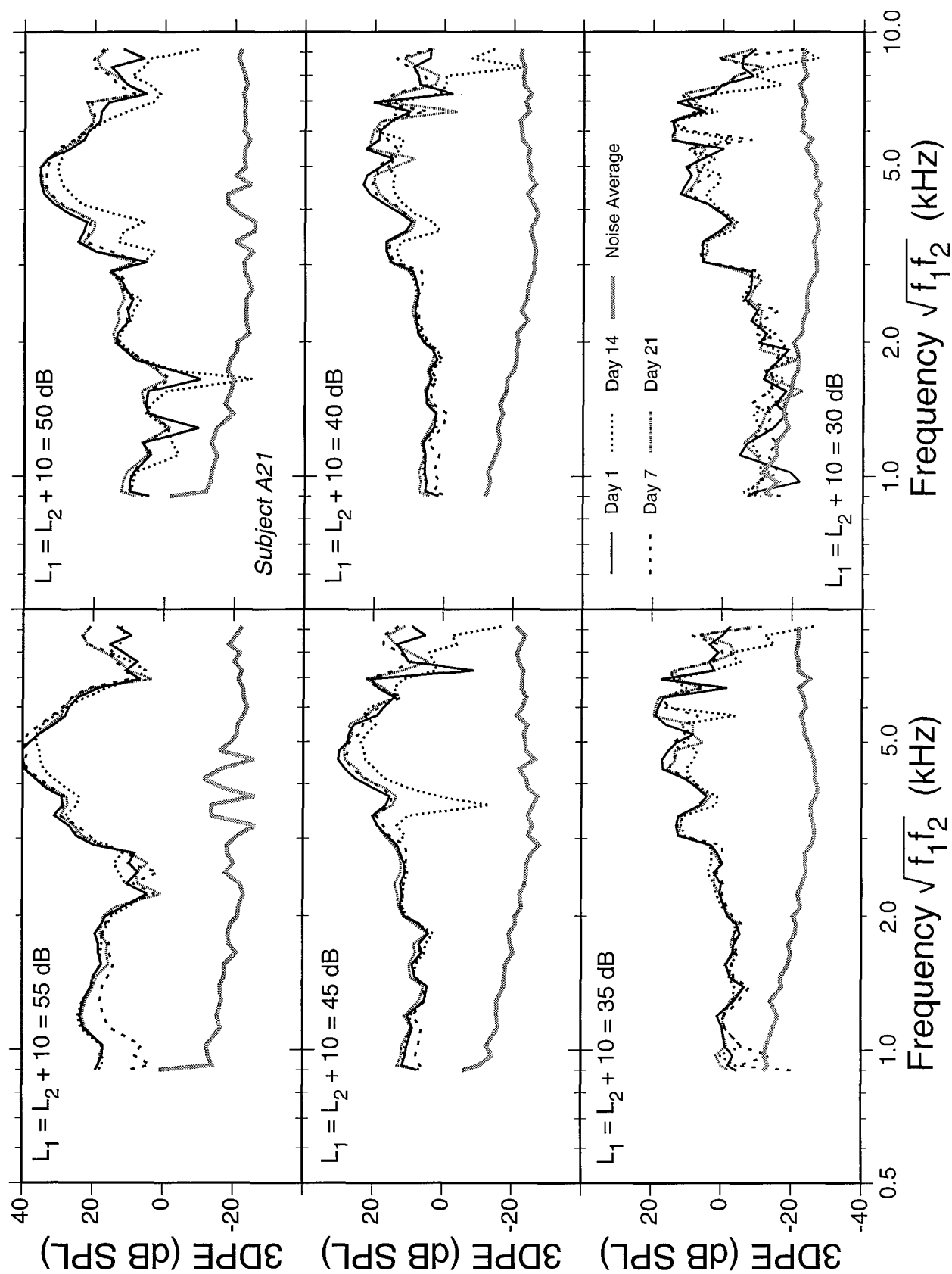
Group mean DPEgrams measured at various times after noise exposure were presented at the beginning of this data appendix (Pages 74 and 75). The final set of figures (Pages 131 through 150) show the individual pre- and postexposure DPEgrams measured 1, 7, 14, and 21 days after noise exposure using the indicated equal and unequal primary levels. Each DPEgram represents the results of a single measurement across the primary frequency ranges. The solid lines represent the individual subject DPEgrams measured one day following noise exposure and the various dotted lines show the individual subject DPEgrams in successive tests as indicated in the legend. The thick gray line represents the average noise floor over the four postexposure measurements.

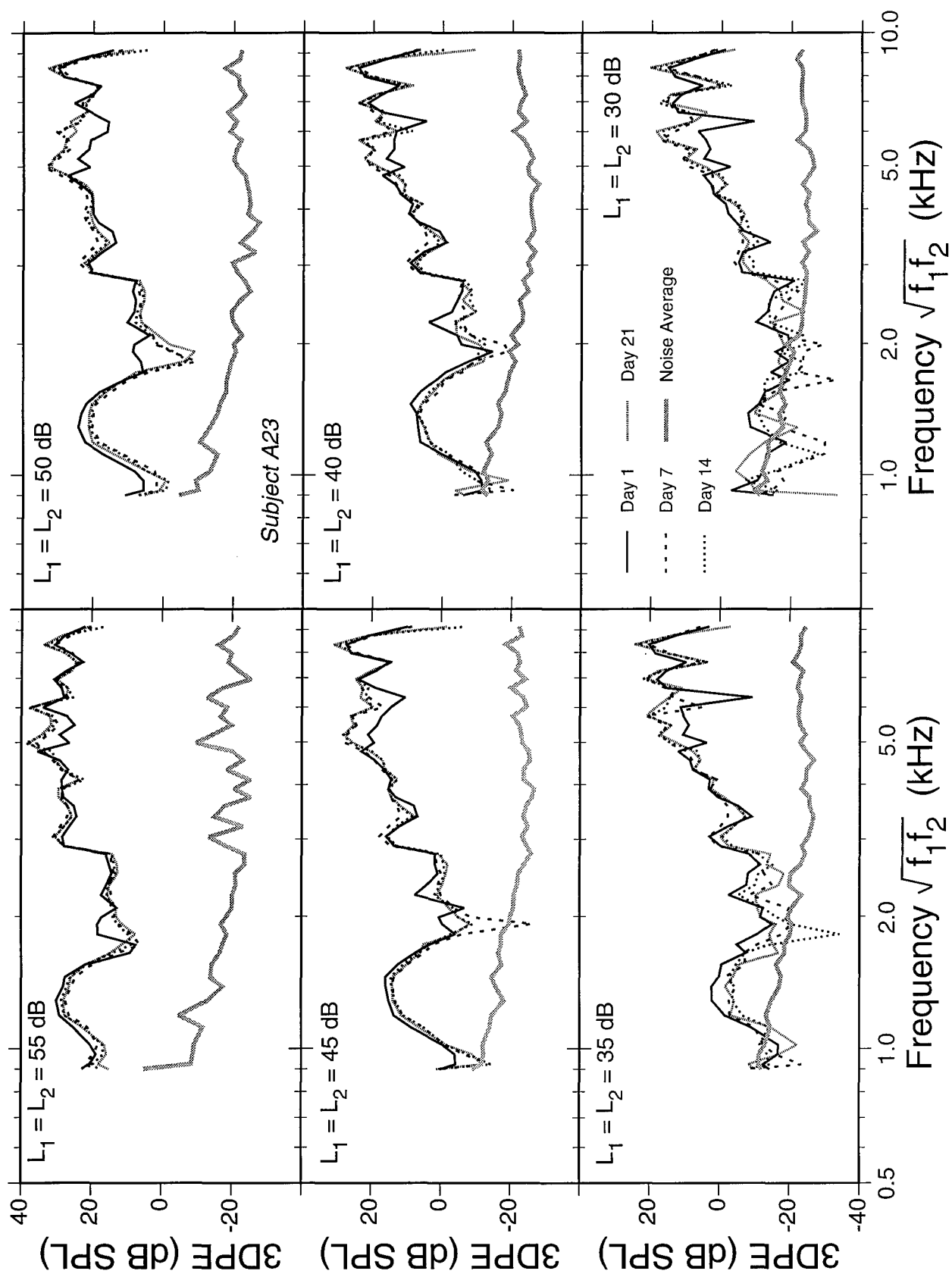


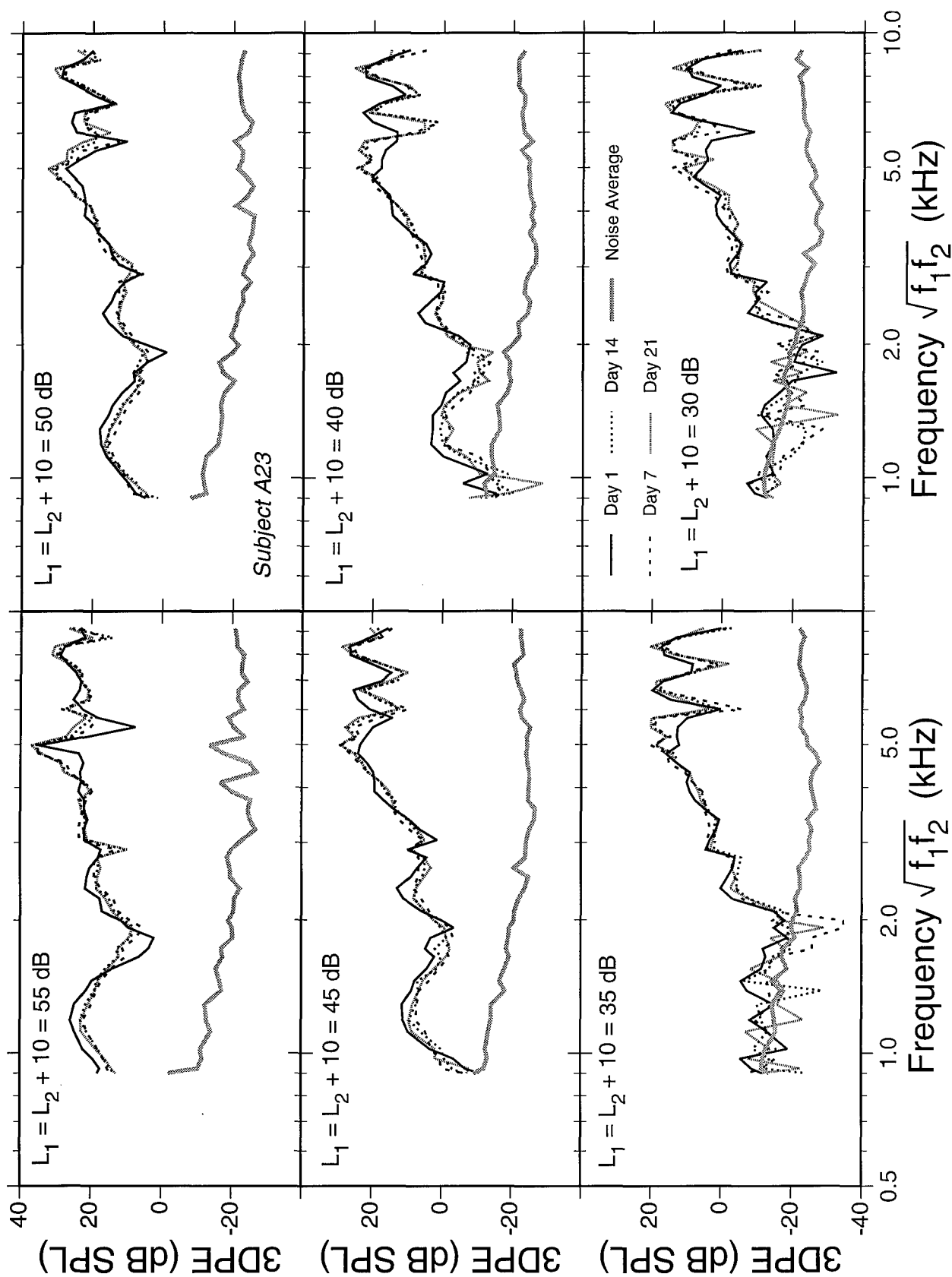


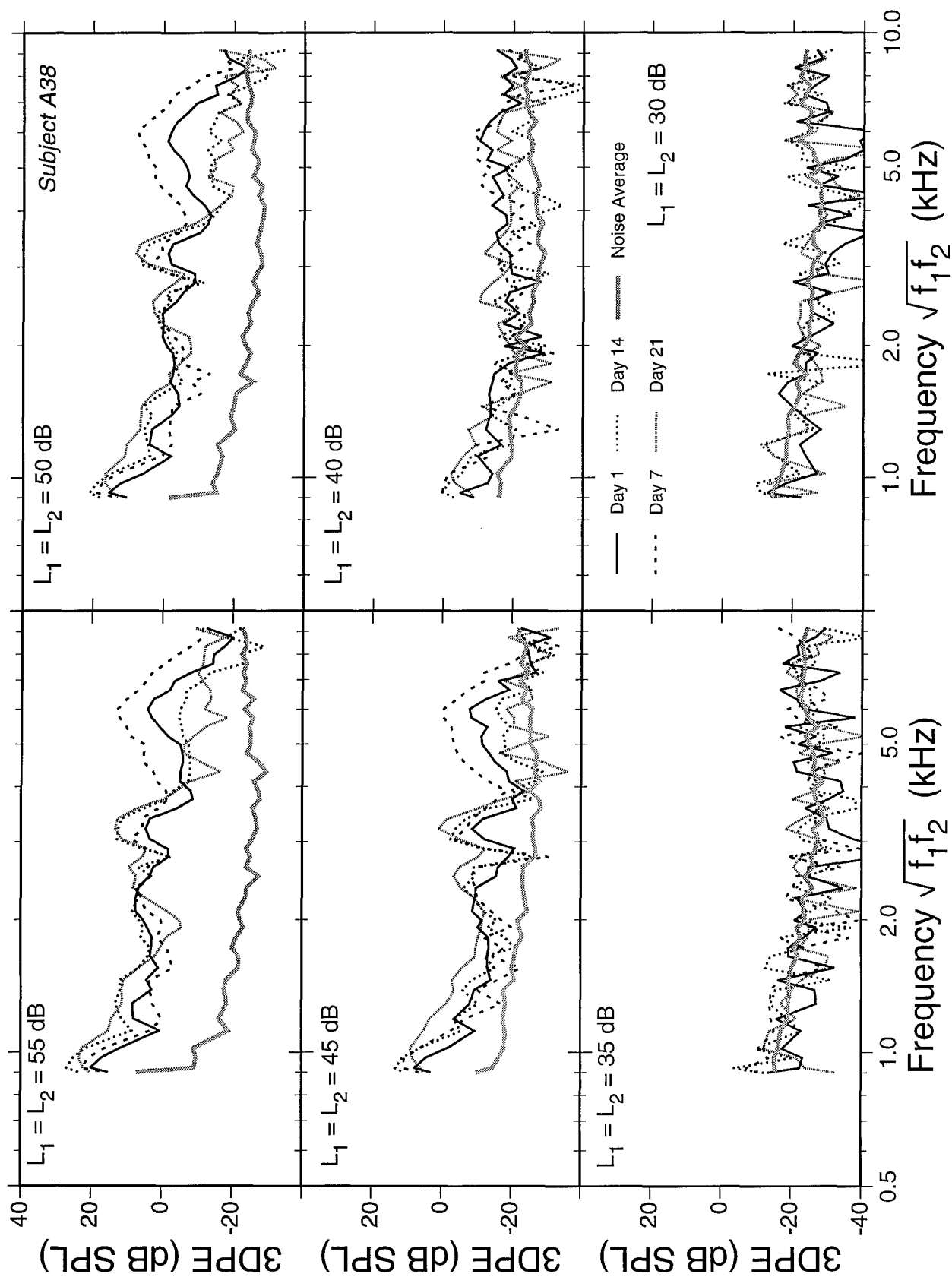


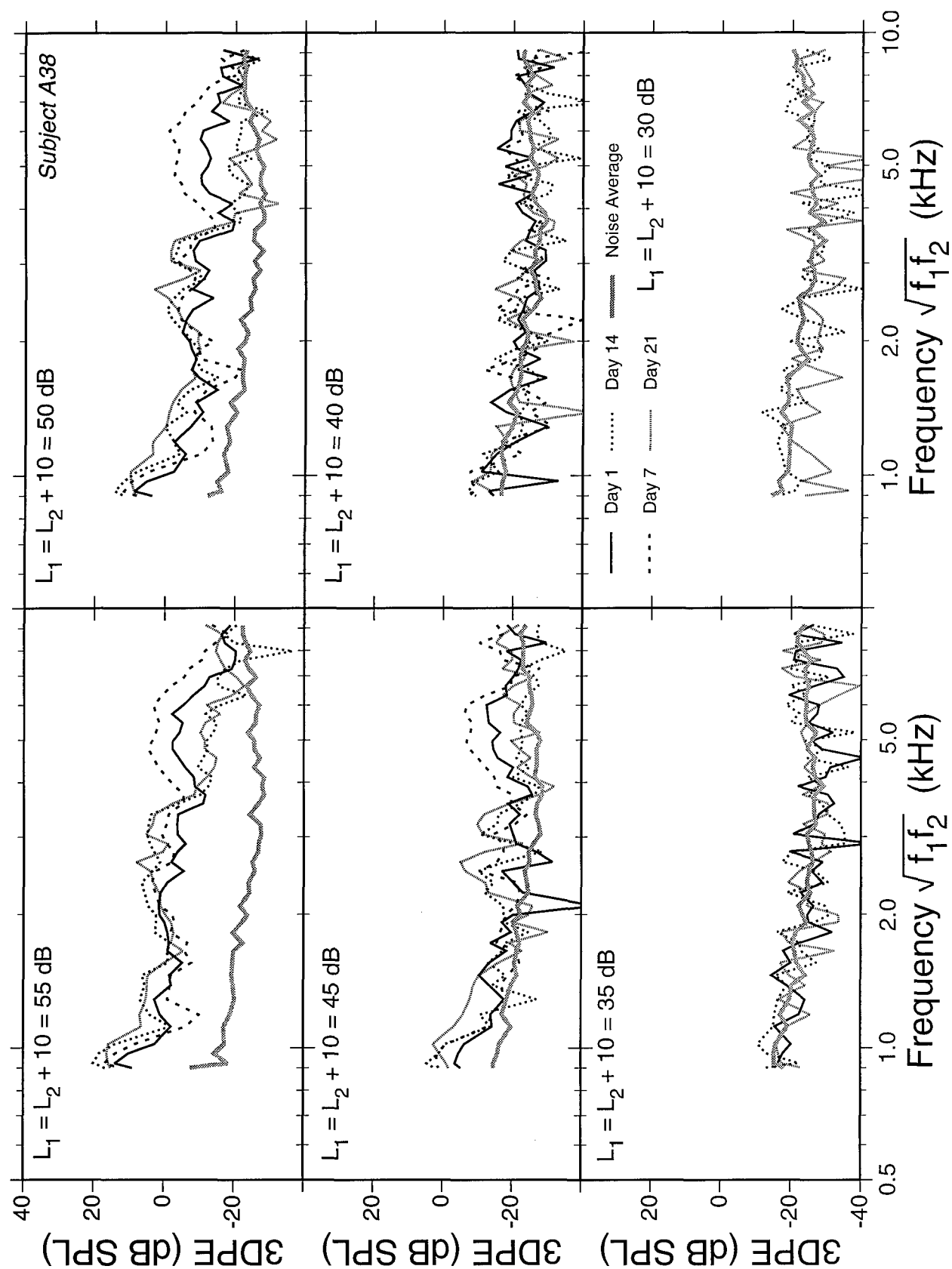


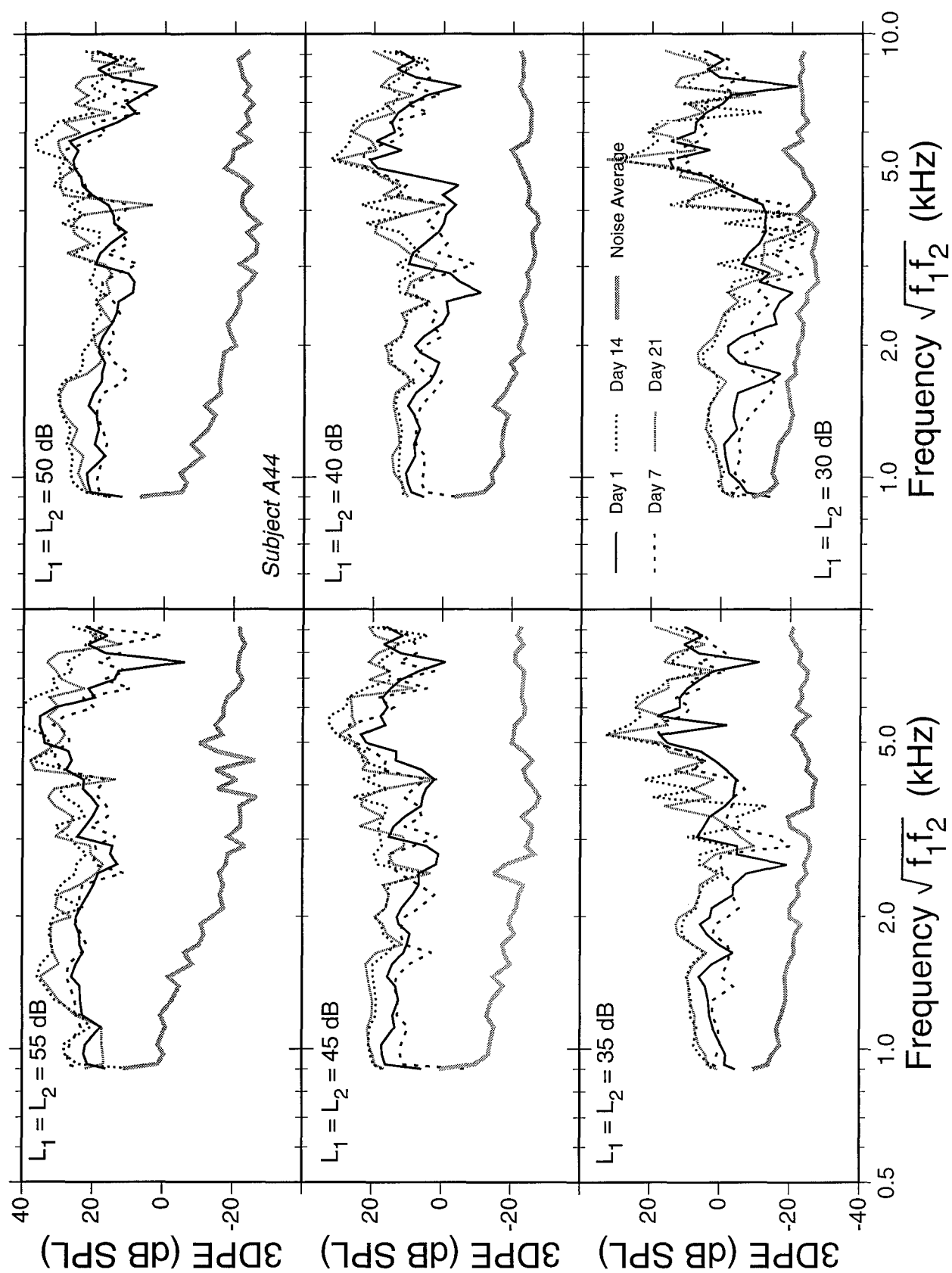




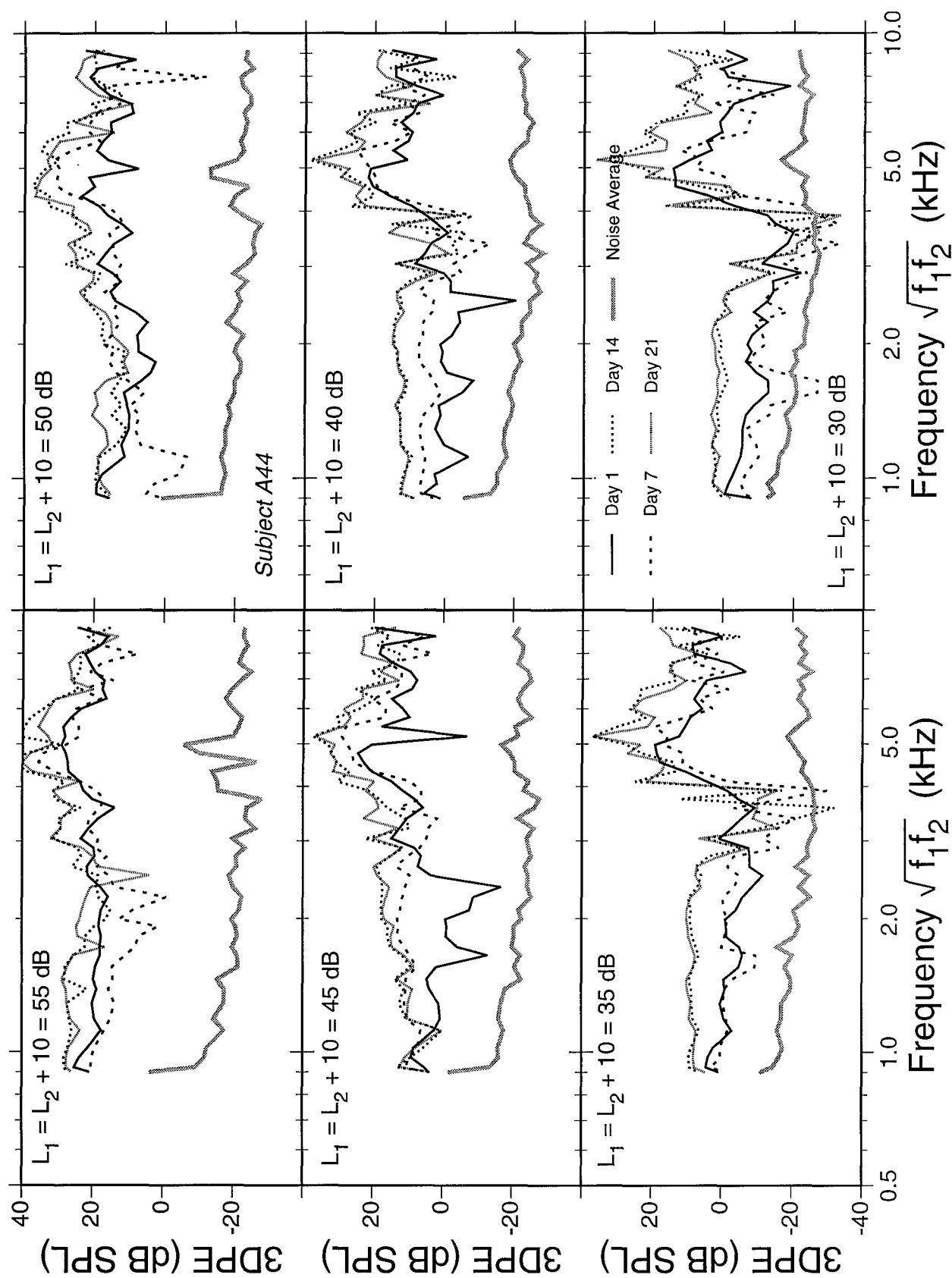


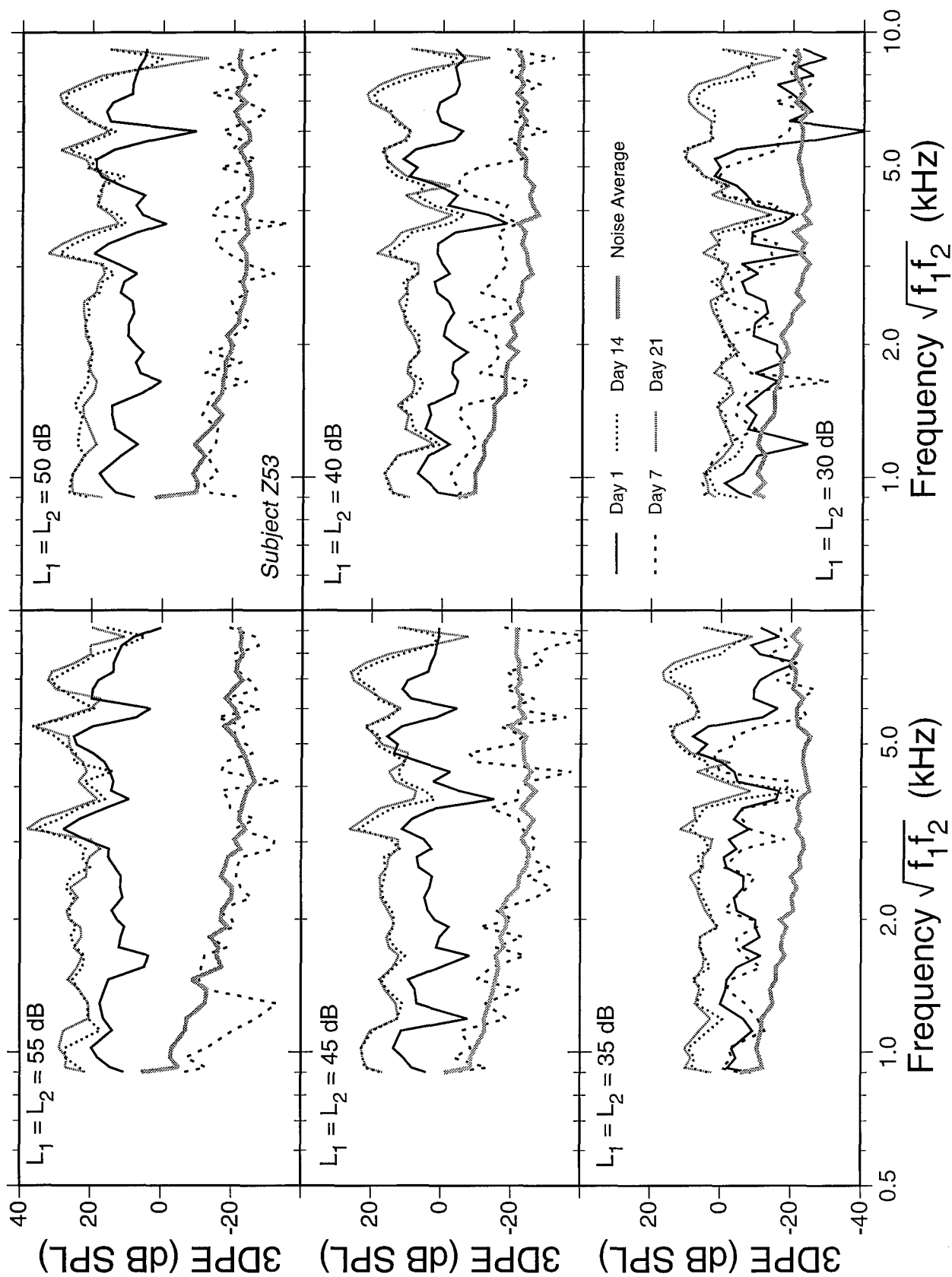


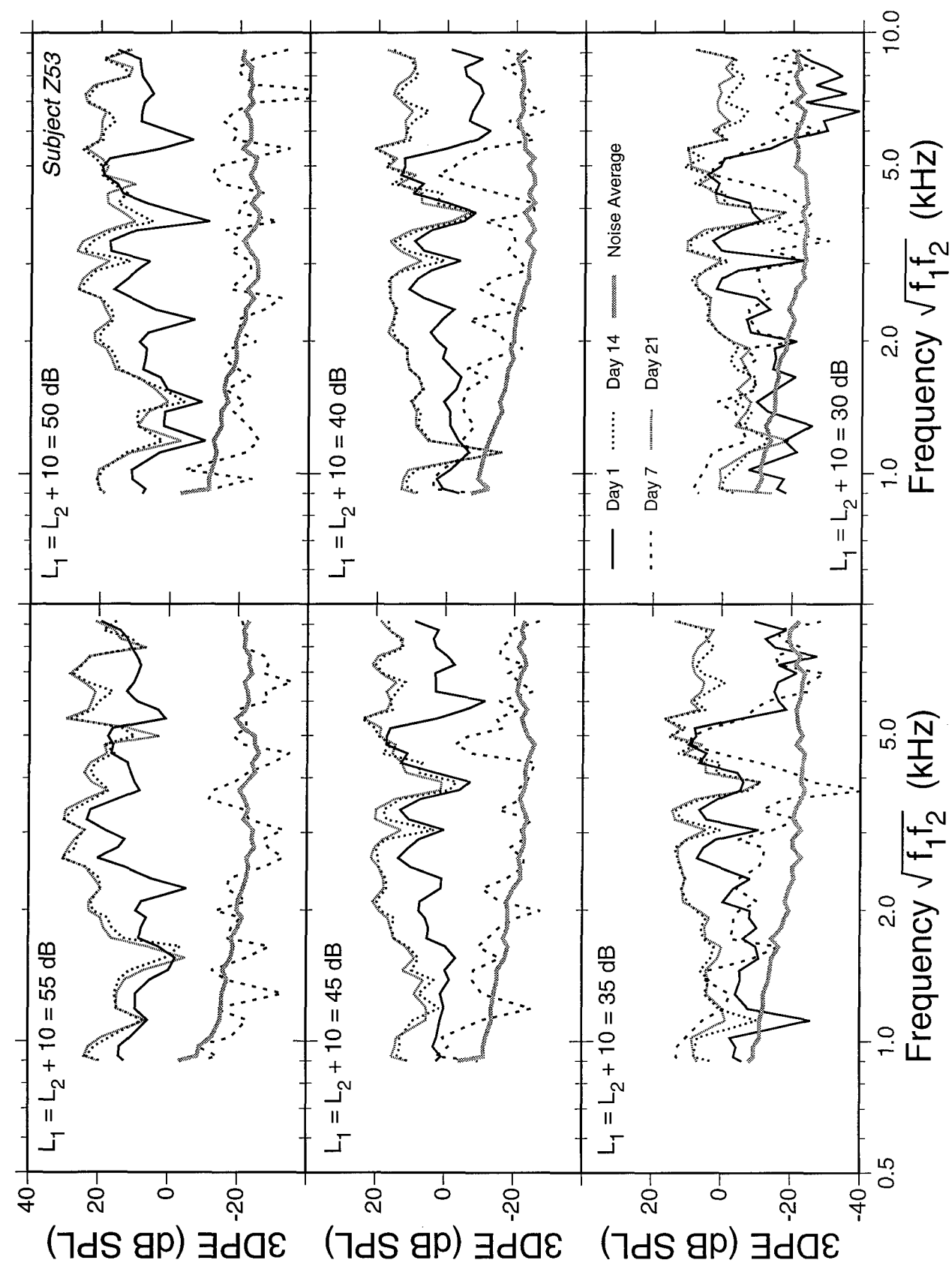


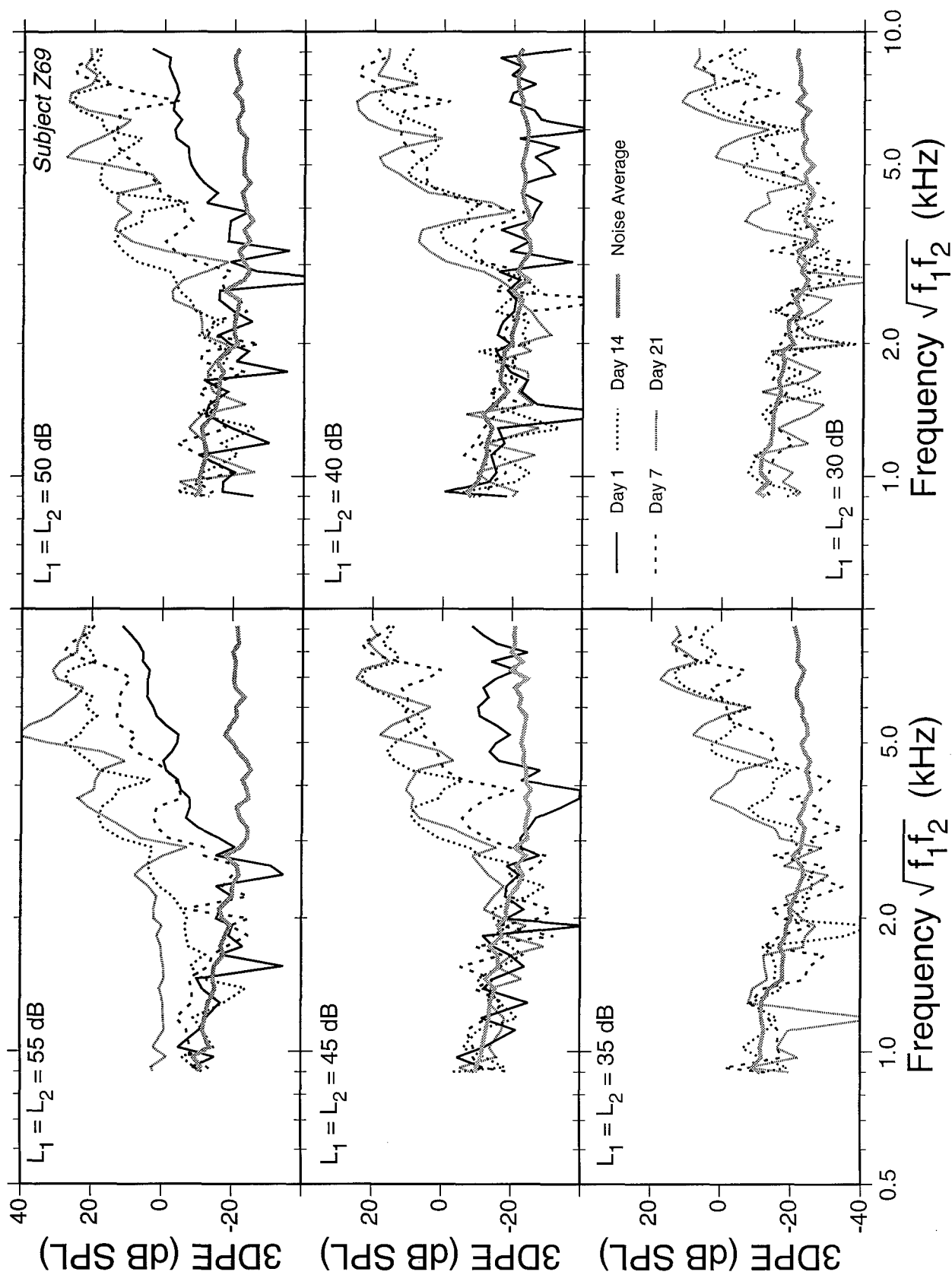


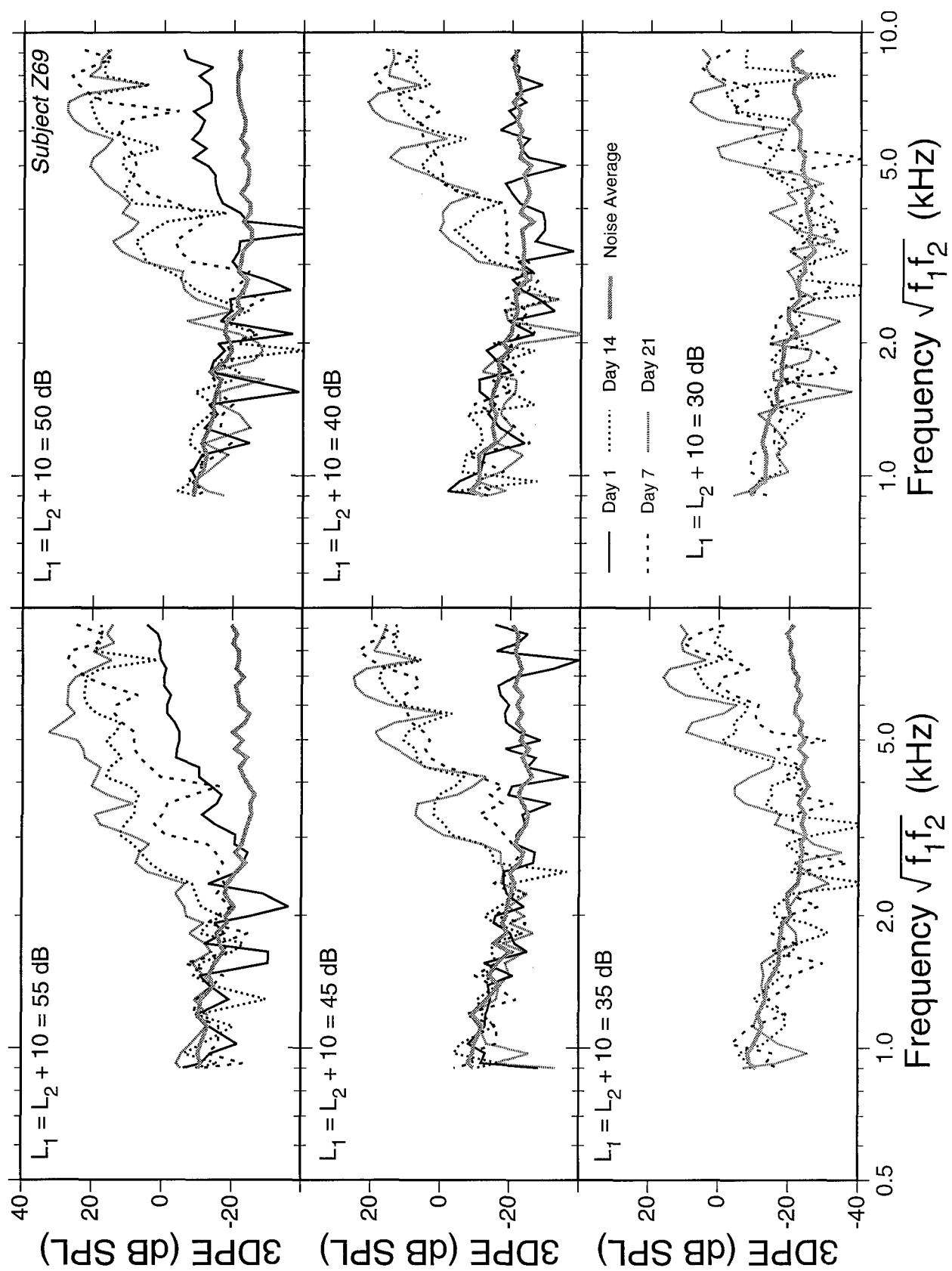


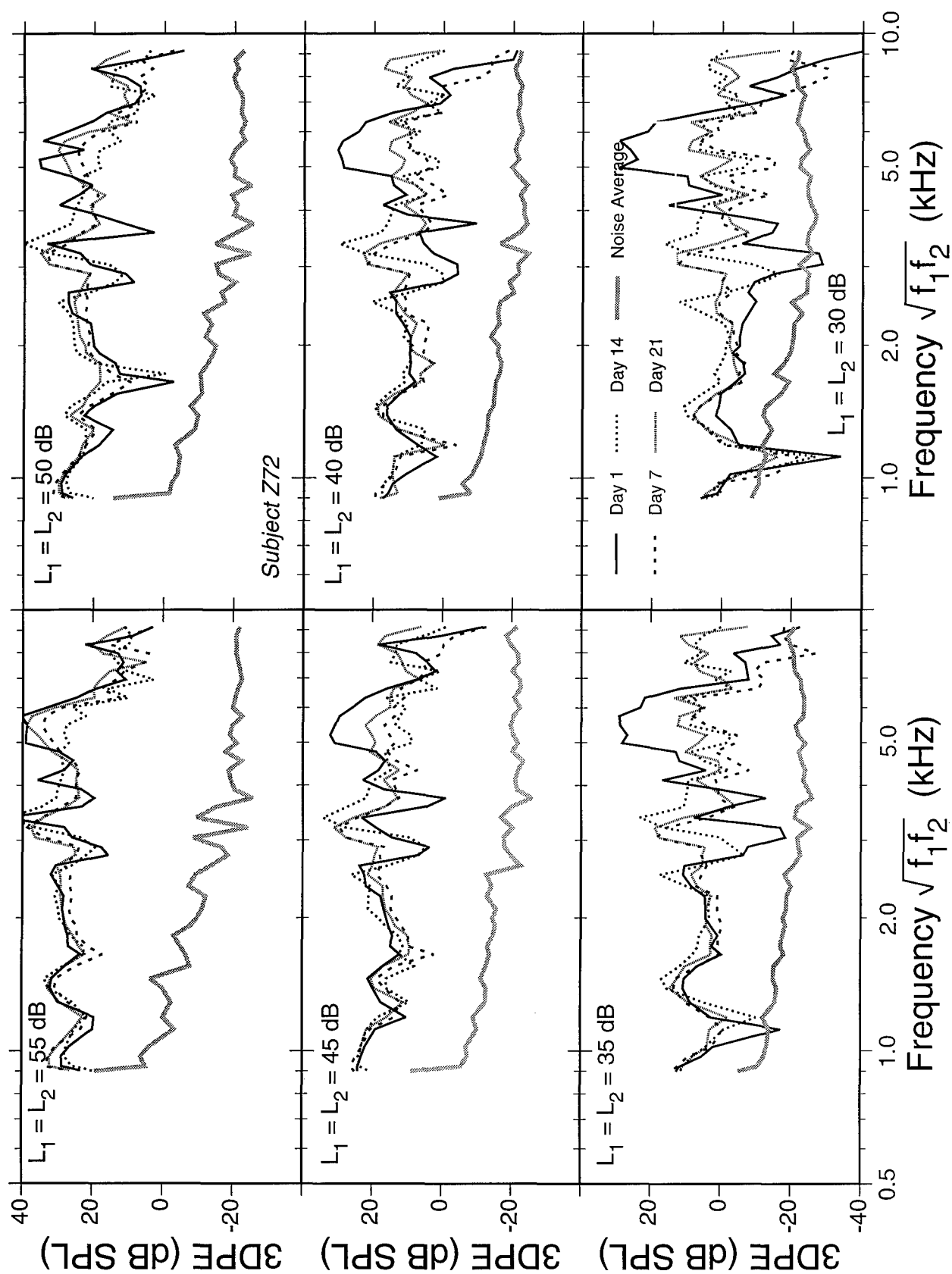


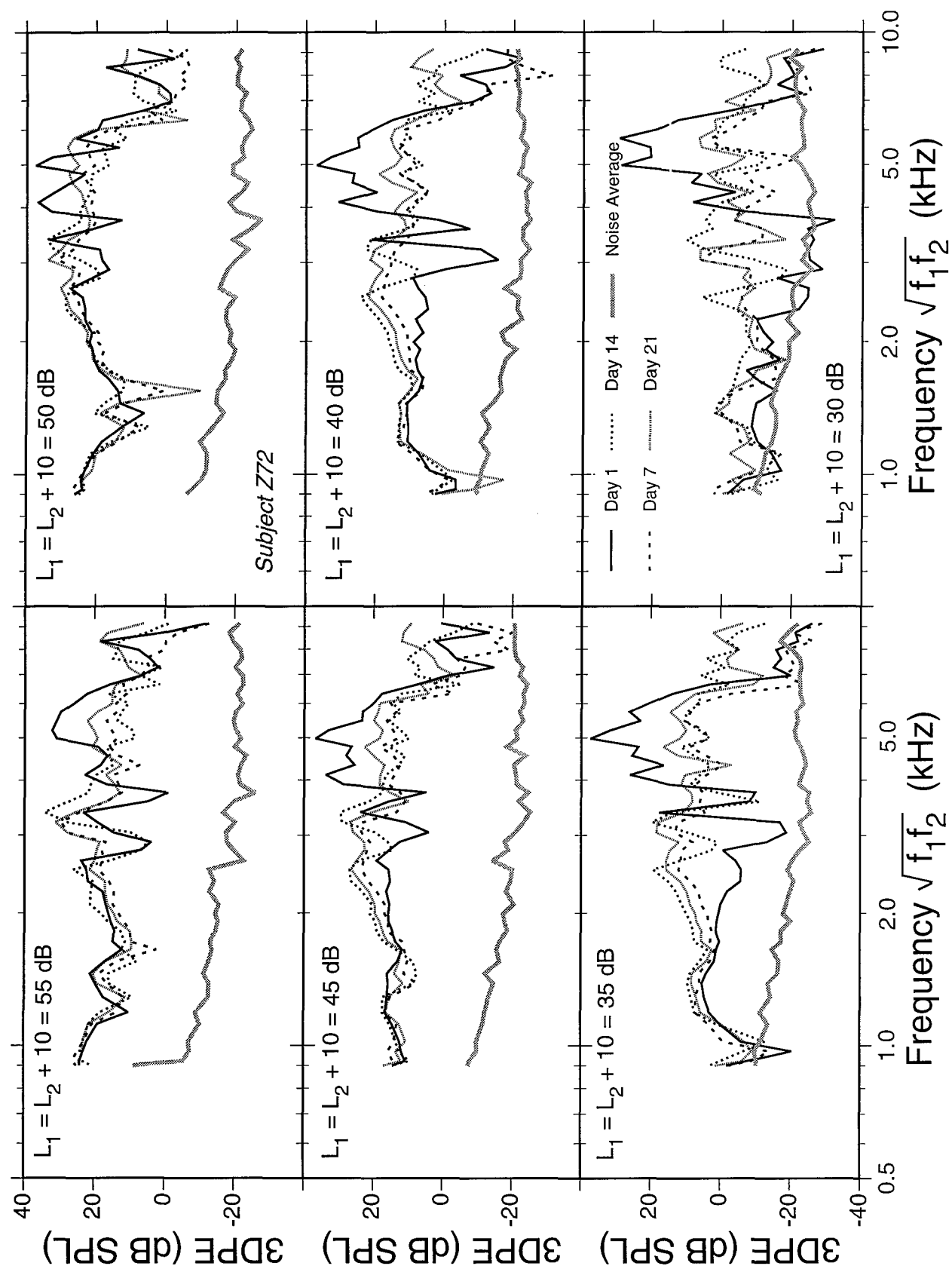


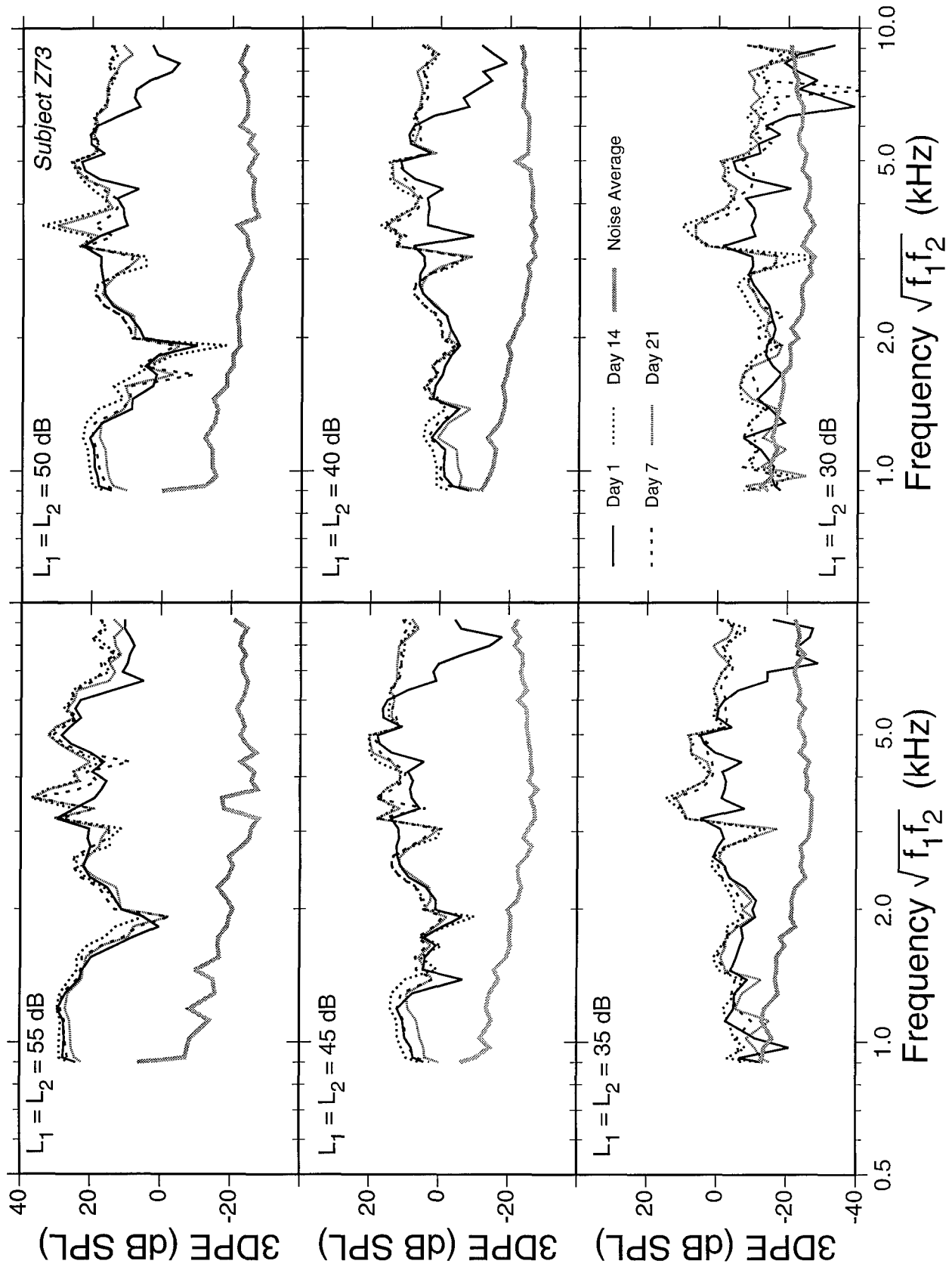




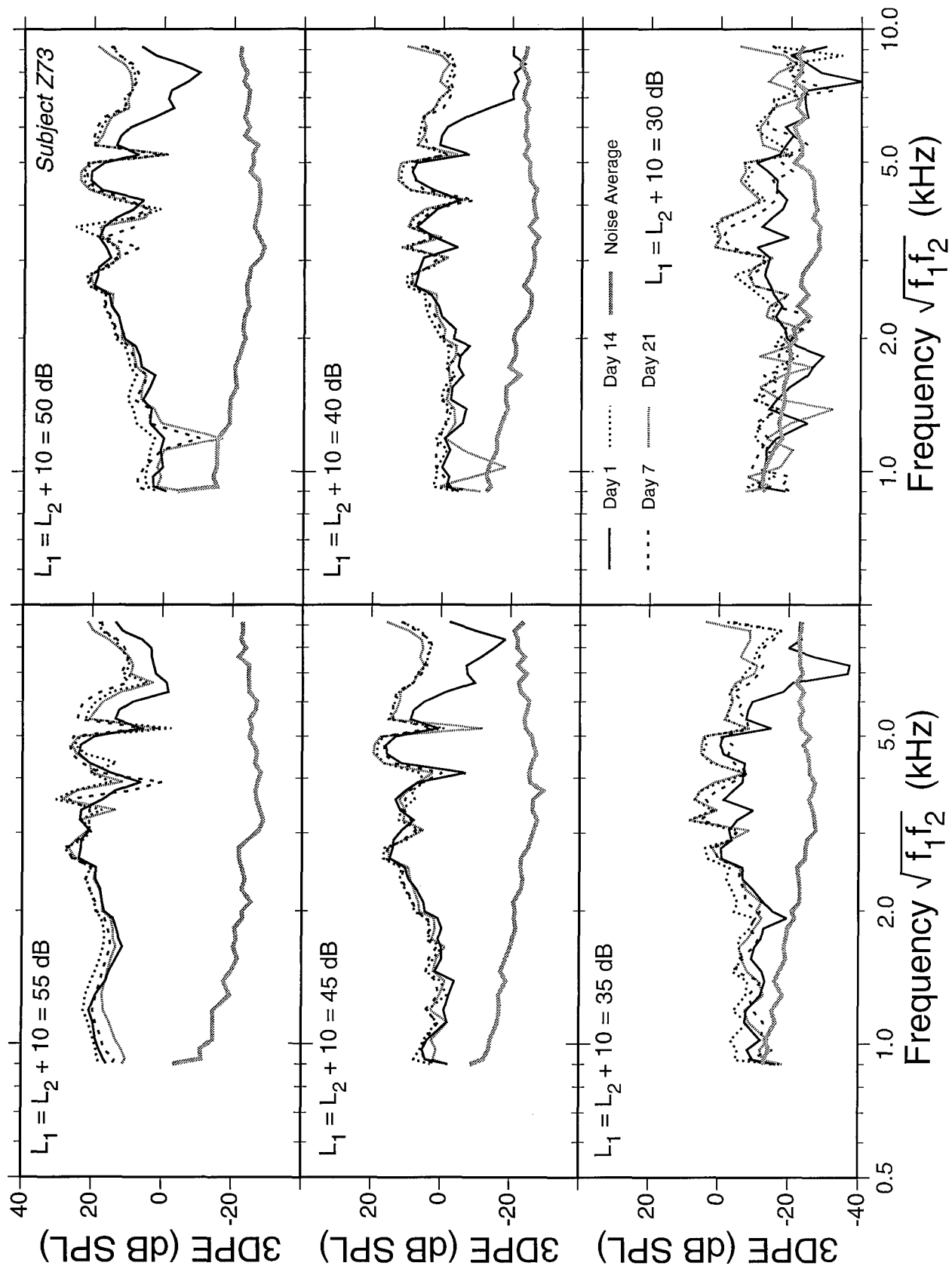


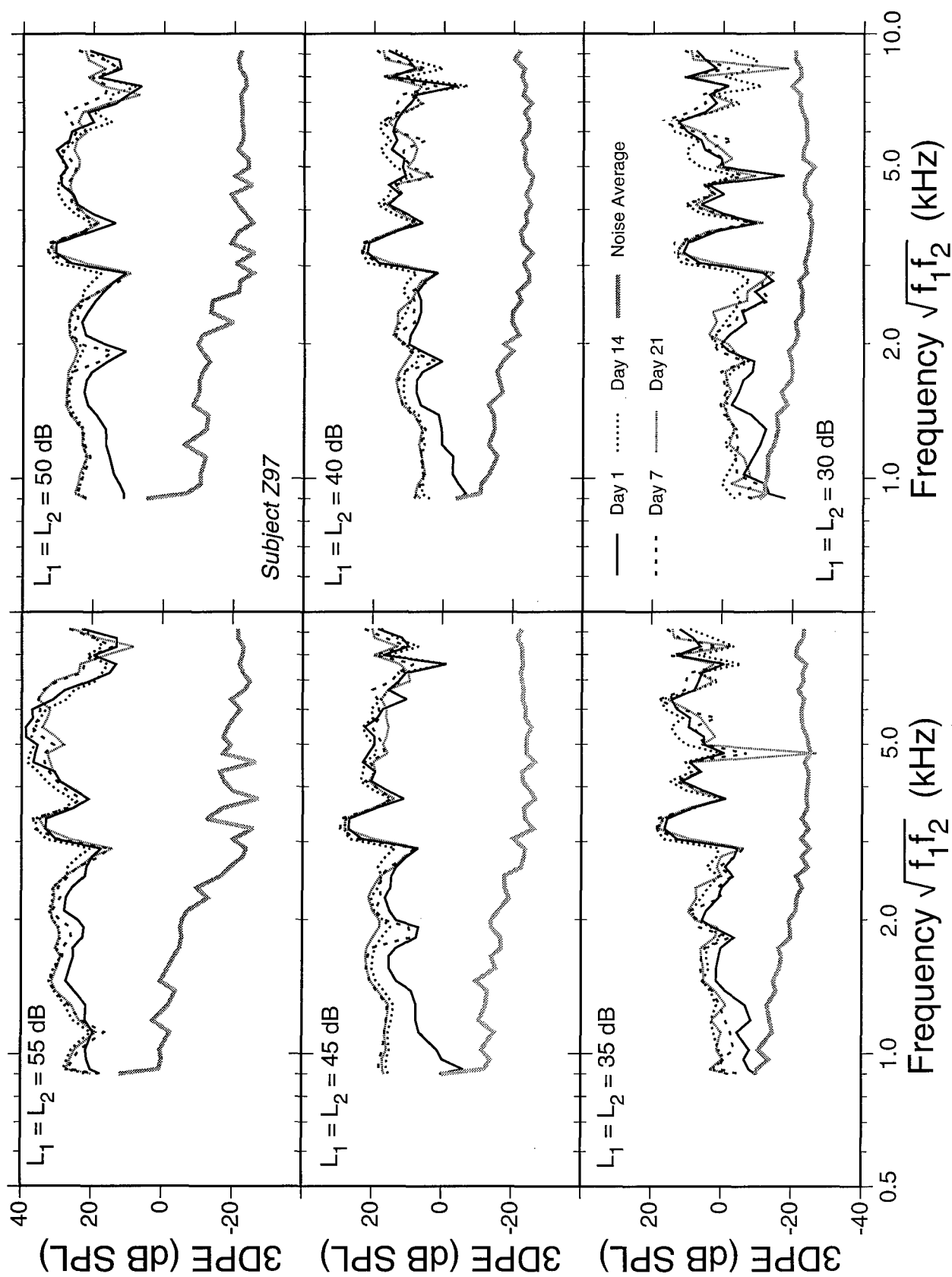


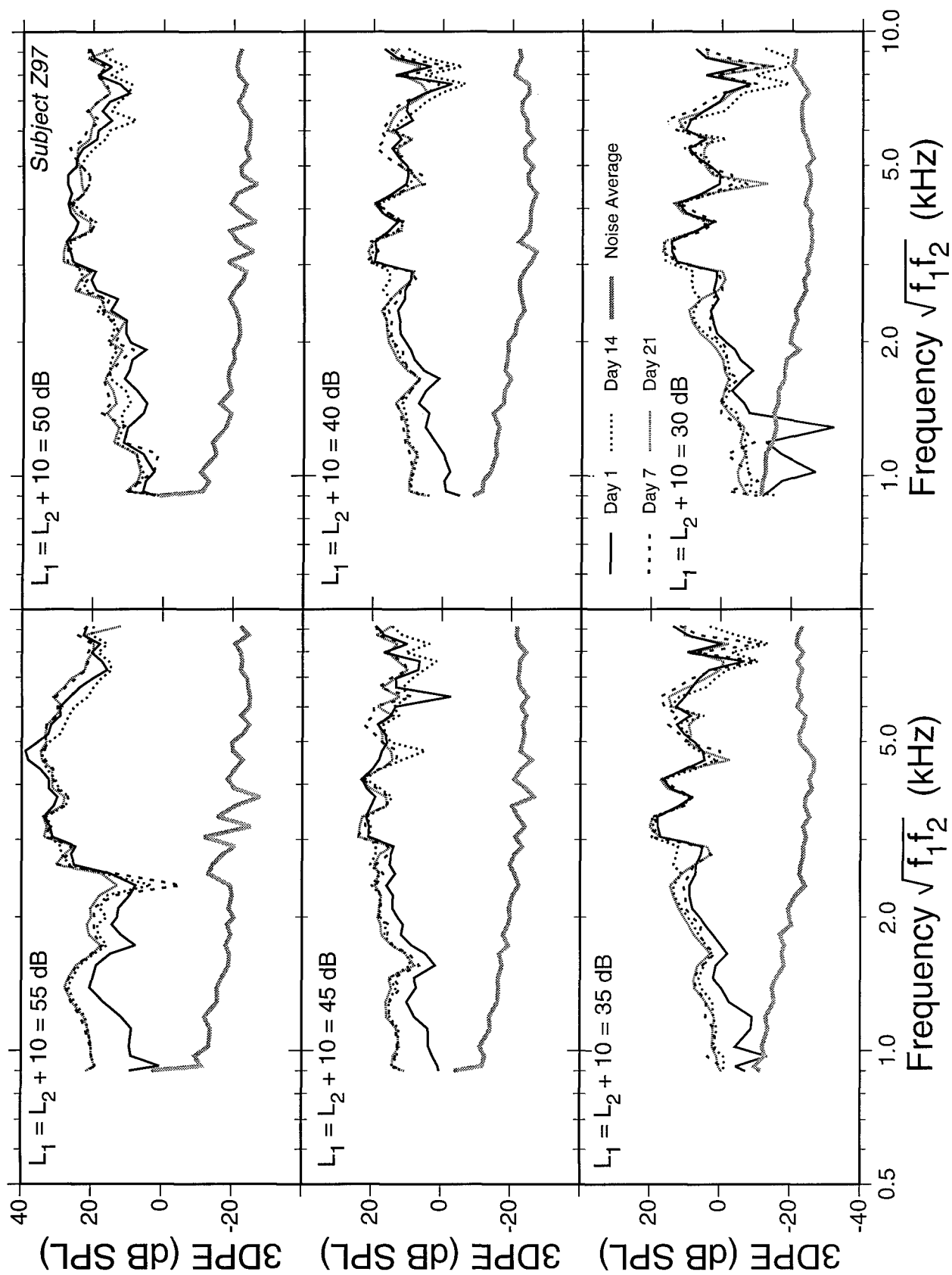












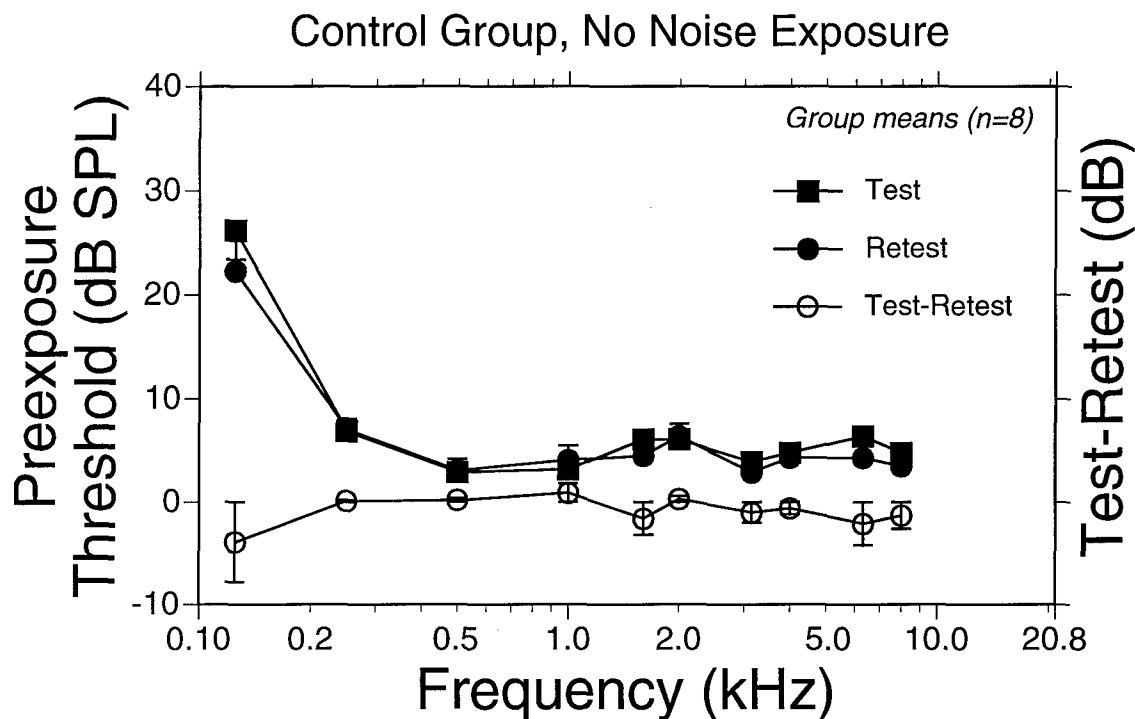
## APPENDIX B

### Summary Data for the Control Group

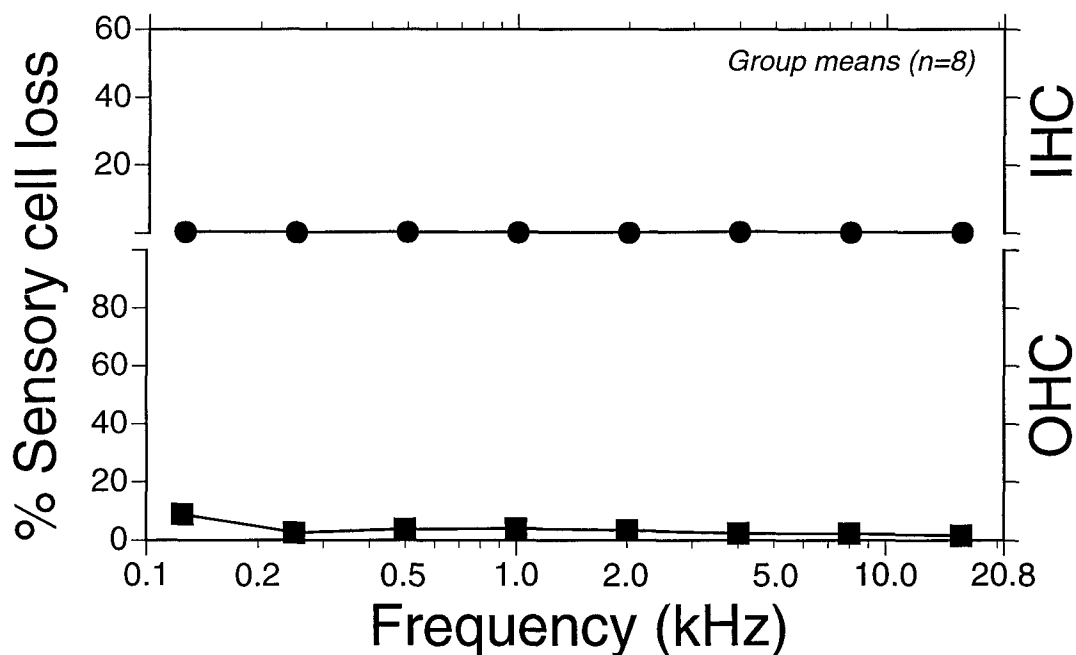
#### Animal #

A41	-	Completed the Entire Protocol
A52	-	Completed the Entire Protocol
A61	-	Completed the Entire Protocol
A89	-	Completed the Entire Protocol
B17	-	Completed the Entire Protocol
B84	-	Completed the Audiometry Protocol Only
B86	-	Completed the Entire Protocol
B96	-	Completed the Entire Protocol
Z87	-	Completed the Entire Protocol

Includes audiometric thresholds, otoacoustic emissions,  
and histology.



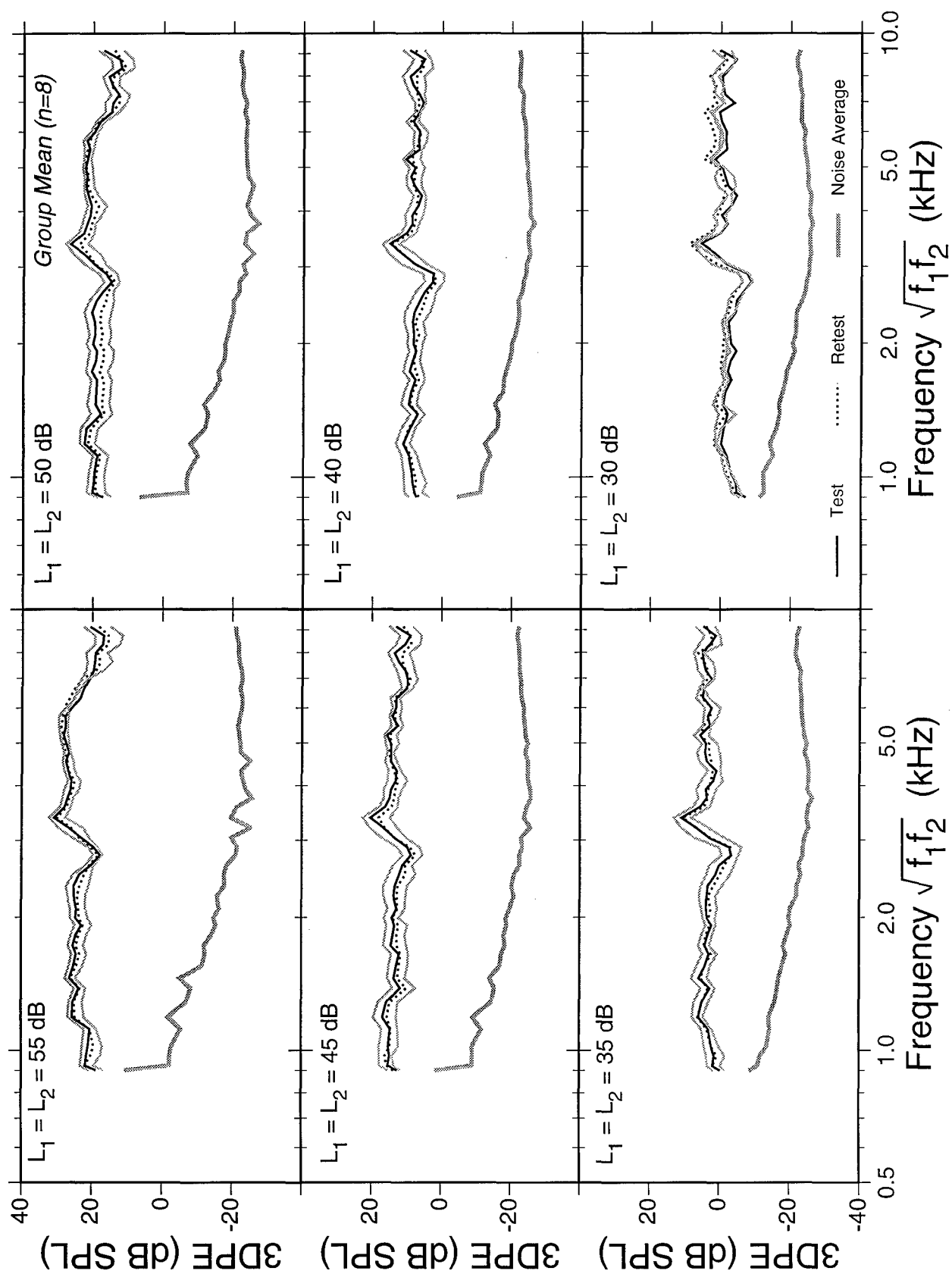
Group thresholds (test-■ and retest-●) and permanent threshold shifts (○) from a group of animals which were not exposed to noise. Error bars represent one standard error of the mean.

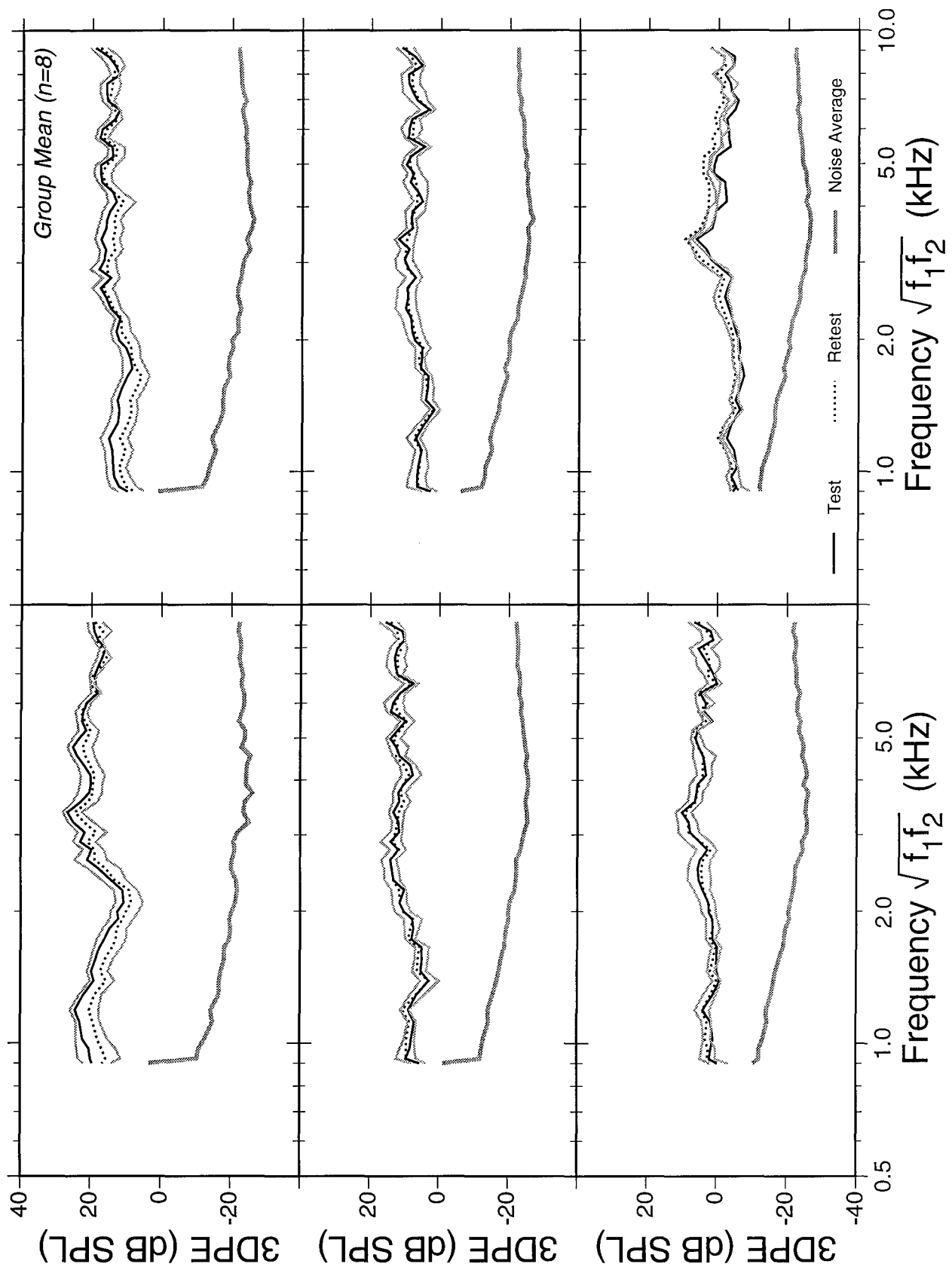


Group mean percent inner (●) and (■) outer hair cell loss from a group of animals which were not exposed to noise. Error bars represent one standard error of the mean.

## Group Mean DPEgrams

Group mean DPEgrams (Pages 154 and 155) at the indicated primary levels for a group of 8 animals that were not exposed to noise. The solid lines represent the mean of the first set of DPEgrams at the six equal-primary measurements ( $L_1 = L_2 = 55$  to 30 dB SPL) and six unequal-primary ( $L_1 = L_2 + 10 = 55$  to 30 dB SPL) measurements. Each set of DPEgrams for each subject represents the average of three measurements made on different days. The dashed lines represent the mean measurements for the second set of three DPEgrams made at least one week after the first measurements. The upper gray line represents one standard error of the mean above the first set of measurements and the lower gray line represents one standard error of the mean below the second set of measurements. The thick gray line represents the average noise floor over the six measurements.







## Group Mean and Individual Audiometry

The next tables (Pages 157 through 162) present the group summary data (means and standard errors of the mean) and individual audiometric data. The measurements specified as "Test" are the average of five threshold determinations made on different days. Thresholds were measured again ("Retest") five times on different days at each test frequency at least seven days following the first series of tests.

# Group Means

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	26.2	6.9	2.9	3.2	6.1	6.1	3.9	4.8	6.4	4.8
Retest Thresholds (dB SPL)										
Retest 1	22.5	9.2	4.7	6.4	6.4	6.4	5.3	4.7	8.6	2.5
Retest 2	21.4	6.4	4.7	4.2	3.1	5.3	4.7	8.6	5.3	2.5
Retest 3	21.4	8.6	3.6	5.8	3.1	7.5	-1.4	1.9	1.4	2.5
Retest 4	23.1	4.7	-0.3	1.9	5.8	6.9	5.8	4.2	5.3	7.5
Retest 5	23.1	6.4	2.5	1.9	4.2	5.8	0.3	1.9	0.8	2.5
Retest Mean	22.3	7.1	3.1	4.1	4.5	6.4	2.9	4.3	4.3	3.5
Test-Retest	-3.9	0.1	0.1	0.9	-1.6	0.3	-1.0	-0.6	-2.1	-1.3

# Group Standard Errors

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	1.1	1.0	1.1	1.4	1.2	1.2	1.3	1.0	1.5	0.7
Retest 1	2.5	2.8	2.6	3.0	2.9	3.0	3.0	2.5	1.6	2.6
Retest 2	2.9	2.3	2.2	2.6	2.4	3.3	3.1	2.6	2.5	2.8
Retest 3	2.6	3.0	3.0	2.2	2.3	2.5	2.9	2.4	3.2	2.0
Retest 4	1.9	3.6	2.4	1.8	2.4	3.8	3.6	2.2	3.5	1.4
Retest 5	1.9	2.3	2.9	3.4	3.0	2.0	2.6	2.6	2.5	1.4
Retest Mean	1.4	1.6	1.3	1.4	1.5	2.0	1.9	0.8	1.8	1.1
Test-Retest	1.1	2.0	1.9	2.0	2.0	1.9	2.6	0.9	1.5	1.2

**Subject A41**

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	27.5	4.5	-4.5	1.5	7.5	8.5	5.5	7.5	3.5	4.5
Retest				Retest Thresholds (dB SPL)						
Retest 1	17.5	12.5	12.5	12.5	7.5	22.5	12.5	22.5	7.5	-2.5
Retest 2	27.5	17.5	12.5	7.5	2.5	17.5	12.5	12.5	2.5	2.5
Retest 3	32.5	22.5	12.5	7.5	7.5	17.5	12.5	-2.5	7.5	2.5
Retest 4	32.5	22.5	2.5	7.5	17.5	32.5	27.5	17.5	17.5	2.5
Retest 5	22.5	17.5	2.5	12.5	7.5	12.5	2.5	-2.5	-2.5	7.5
Retest Mean	26.5	18.5	8.5	9.5	8.5	20.5	13.5	9.5	6.5	2.5
Test-Retest	-1.0	14.0	13.0	8.0	1.0	12.0	8.0	2.0	3.0	-2.0

**Subject A52**

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	27.5	10.5	4.5	-5.5	7.5	9.5	-2.5	0.5	8.5	2.5
Retest				Retest Thresholds (dB SPL)						
Retest 1	27.5	12.5	-7.5	17.5	-7.5	17.5	17.5	7.5	12.5	7.5
Retest 2	22.5	2.5	7.5	7.5	7.5	-7.5	2.5	2.5	7.5	7.5
Retest 3	32.5	12.5	12.5	12.5	-2.5	12.5	-7.5	2.5	12.5	-2.5
Retest 4	17.5	2.5	2.5	7.5	12.5	2.5	7.5	2.5	12.5	2.5
Retest 5	22.5	-7.5	-7.5	-7.5	2.5	2.5	7.5	-2.5	2.5	-2.5
Retest Mean	24.5	4.5	1.5	7.5	2.5	5.5	5.5	2.5	9.5	2.5
Test-Retest	-3.0	-6.0	-3.0	13.0	-5.0	-4.0	8.0	2.0	1.0	0.0

**Subject A61**

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	30.5	9.5	2.5	8.5	6.5	11.5	7.5	7.5	5.5	8.5
Retest Thresholds (dB SPL)										
Retest 1	22.5	12.5	12.5	7.5	12.5	7.5	12.5	7.5	7.5	7.5
Retest 2	22.5	2.5	-2.5	2.5	2.5	2.5	12.5	2.5	2.5	7.5
Retest 3	22.5	7.5	2.5	7.5	2.5	2.5	-2.5	2.5	7.5	12.5
Retest 4	22.5	2.5	7.5	2.5	7.5	-2.5	12.5	2.5	12.5	7.5
Retest 5	22.5	7.5	2.5	2.5	-7.5	12.5	-7.5	7.5	7.5	2.5
Retest Mean	22.5	6.5	4.5	4.5	3.5	4.5	5.5	4.5	7.5	7.5
Test-Retest	-8.0	-3.0	2.0	-4.0	-3.0	-7.0	-2.0	-3.0	2.0	-1.0

**Subject A89**

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	22.5	6.5	1.5	0.5	2.5	0.5	8.5	2.5	3.5	6.5
Retest Thresholds (dB SPL)										
Retest 1	12.5	7.5	-7.5	-7.5	-2.5	2.5	2.5	2.5	12.5	-2.5
Retest 2	2.5	-2.5	2.5	-12.5	12.5	7.5	12.5	27.5	7.5	7.5
Retest 3	22.5	-7.5	-7.5	12.5	-7.5	2.5	-12.5	-2.5	-12.5	7.5
Retest 4	12.5	12.5	-7.5	-2.5	-7.5	7.5	2.5	-7.5	12.5	12.5
Retest 5	12.5	7.5	-2.5	-7.5	-7.5	-2.5	-7.5	2.5	2.5	2.5
Retest Mean	12.5	3.5	-4.5	-3.5	-2.5	3.5	-0.5	4.5	4.5	5.5
Test-Retest	-10.0	-3.0	-6.0	-4.0	-5.0	3.0	-9.0	2.0	1.0	-1.0

**Subject B17**

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	25.5	5.5	3.5	4.5	5.5	2.5	0.5	7.5	6.5	4.5
Retest Thresholds (dB SPL)										
Retest 1	32.5	7.5	7.5	2.5	12.5	7.5	7.5	2.5	7.5	-2.5
Retest 2	12.5	2.5	7.5	7.5	2.5	12.5	7.5	2.5	7.5	7.5
Retest 3	22.5	17.5	-7.5	2.5	-2.5	-7.5	-7.5	2.5	7.5	-2.5
Retest 4	22.5	2.5	2.5	2.5	7.5	7.5	2.5	7.5	2.5	7.5
Retest 5	22.5	7.5	12.5	-7.5	2.5	-2.5	-2.5	7.5	-7.5	2.5
Retest Mean	22.5	7.5	4.5	1.5	4.5	3.5	1.5	4.5	3.5	2.5
Test-Retest	-3.0	2.0	1.0	-3.0	-1.0	1.0	1.0	-3.0	-3.0	-2.0

**Subject B84**

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	31.5	7.5	3.5	3.5	8.5	6.5	1.5	7.5	13.5	1.5
Retest Thresholds (dB SPL)										
Retest 1	32.5	-7.5	2.5	12.5	17.5	-2.5	7.5	2.5	12.5	12.5
Retest 2	27.5	7.5	-7.5	-2.5	12.5	-12.5	-7.5	7.5	7.5	12.5
Retest 3	12.5	7.5	7.5	-7.5	12.5	12.5	7.5	12.5	7.5	2.5
Retest 4	22.5	2.5	2.5	7.5	2.5	7.5	12.5	7.5	-12.5	2.5
Retest 5	32.5	2.5	7.5	2.5	12.5	7.5	7.5	-12.5	2.5	-2.5
Retest Mean	25.5	2.5	2.5	2.5	11.5	2.5	5.5	3.5	3.5	5.5
Test-Retest	-6.0	-5.0	-1.0	-1.0	3.0	-4.0	4.0	-4.0	-10.0	4.0

**Subject B86**

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	23.5	10.5	6.5	3.5	8.5	5.5	8.5	3.5	0.5	5.5
Retest Thresholds (dB SPL)										
Retest 1	22.5	22.5	12.5	-7.5	-2.5	-2.5	-12.5	-2.5	7.5	-12.5
Retest 2	27.5	7.5	7.5	12.5	-7.5	12.5	-12.5	7.5	-12.5	-12.5
Retest 3	12.5	12.5	-7.5	2.5	2.5	7.5	-7.5	7.5	-7.5	-7.5
Retest 4	27.5	-17.5	7.5	-2.5	2.5	2.5	-7.5	2.5	-7.5	7.5
Retest 5	27.5	12.5	12.5	-2.5	17.5	2.5	7.5	12.5	-12.5	7.5
Retest Mean	23.5	7.5	6.5	0.5	2.5	4.5	-6.5	5.5	-6.5	-3.5
Test-Retest	0.0	-3.0	0.0	-3.0	-6.0	-1.0	-15.0	2.0	-7.0	-9.0

**Subject B96**

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	21.5	1.5	2.5	7.5	-1.5	2.5	2.5	1.5	3.5	3.5
Retest Thresholds (dB SPL)										
Retest 1	12.5	2.5	7.5	12.5	12.5	-2.5	-2.5	-2.5	-2.5	7.5
Retest 2	27.5	2.5	2.5	2.5	2.5	2.5	12.5	7.5	12.5	-2.5
Retest 3	12.5	2.5	12.5	12.5	12.5	7.5	-2.5	-12.5	-12.5	2.5
Retest 4	22.5	2.5	-7.5	-7.5	7.5	-7.5	2.5	2.5	-2.5	12.5
Retest 5	27.5	7.5	7.5	22.5	12.5	7.5	7.5	7.5	2.5	7.5
Retest Mean	20.5	3.5	4.5	8.5	9.5	1.5	3.5	0.5	-0.5	5.5
Test-Retest	-1.0	2.0	2.0	1.0	11.0	-1.0	1.0	-1.0	-4.0	2.0

**Subject Z87**

Frequency (Hz)	125	250	500	1000	1600	2000	3150	4000	6300	8000
Test (dB SPL)	25.5	6.5	6.5	4.5	9.5	7.5	3.5	5.5	12.5	6.5
Retest Thresholds (dB SPL)										
Retest 1	22.5	12.5	2.5	7.5	7.5	7.5	2.5	2.5	12.5	7.5
Retest 2	22.5	17.5	12.5	12.5	-7.5	12.5	2.5	7.5	12.5	-7.5
Retest 3	22.5	2.5	7.5	2.5	2.5	12.5	7.5	7.5	2.5	7.5
Retest 4	27.5	12.5	-12.5	2.5	2.5	12.5	-7.5	2.5	12.5	12.5
Retest 5	17.5	2.5	-12.5	2.5	-2.5	12.5	-12.5	-2.5	12.5	-2.5
Retest Mean	22.5	9.5	-0.5	5.5	0.5	11.5	-1.5	3.5	10.5	3.5
Test-Retest	-3.0	3.0	-7.0	1.0	-9.0	4.0	-5.0	-2.0	-2.0	-3.0

## Individual and Group Mean Histology Summary

Group summary data (means, standard deviations, and standard errors of the mean) are presented for total and percent cell losses measured in octave-band lengths of the cochlea on Pages 164 and 165. Following the summary data, individual animal total and percent cell losses are presented (Pages 166 through 170). Following the tabulated data, individual animal cochleograms are presented on Pages 171 through 178. The three graphs on these pages show: (top) a "standard" cochleogram showing percent inner and outer sensory cell losses with the permanent threshold shift (dB); (middle) percent cell losses in each of the three rows of outer hair cells; and (bottom) percent missing supporting (pillar) cells.



Control Group  
No Noise Exposure

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Group means							
0.125 kHz	0.6	5.5	17.0	26.6	49.1	0.0	0.0
0.25 kHz	0.6	4.3	5.4	16.1	25.8	0.0	0.1
0.5 kHz	0.7	14.4	11.5	11.5	37.4	0.1	0.2
1 kHz	0.6	12.6	17.5	7.6	37.8	0.0	0.0
2 kHz	0.4	17.4	9.9	5.3	32.5	0.0	0.1
4 kHz	1.0	13.1	4.3	5.5	22.9	0.0	0.0
8 kHz	0.6	13.4	5.1	4.0	22.5	0.0	0.0
16 kHz	0.5	7.5	3.4	2.8	13.6	0.0	0.0
TOTALS	5.1	88.1	74.0	79.4	241.5	0.1	0.5
Group standard deviations							
0.125 kHz	1.1	5.1	9.1	16.2	21.9	0.0	0.0
0.25 kHz	1.2	6.6	5.6	9.6	19.3	0.0	0.4
0.5 kHz	1.0	33.0	20.5	9.7	53.7	0.4	0.7
1 kHz	0.9	28.6	38.4	6.3	70.7	0.0	0.0
2 kHz	0.7	28.6	18.1	3.4	46.6	0.0	0.4
4 kHz	1.2	21.6	6.9	6.6	34.4	0.0	0.0
8 kHz	0.7	30.6	9.4	4.4	42.4	0.0	0.0
16 kHz	0.5	15.0	4.4	2.4	20.3	0.0	0.0
TOTALS	4.4	130.1	87.3	33.2	241.5	0.4	1.1
Group standard errors							
0.125 kHz	0.4	1.8	3.2	5.7	7.7	0.0	0.0
0.25 kHz	0.4	2.3	2.0	3.4	6.8	0.0	0.1
0.5 kHz	0.4	11.7	7.2	3.4	19.0	0.1	0.2
1 kHz	0.3	10.1	13.6	2.2	25.0	0.0	0.0
2 kHz	0.3	10.1	6.4	1.2	16.5	0.0	0.1
4 kHz	0.4	7.6	2.5	2.3	12.1	0.0	0.0
8 kHz	0.3	10.8	3.3	1.5	15.0	0.0	0.0
16 kHz	0.2	5.3	1.6	0.9	7.2	0.0	0.0
TOTALS	1.6	46.0	30.9	11.7	85.4	0.1	0.4

Control Group  
No Noise Exposure

Percent sensory cell losses over octave band frequencies

		1st row	2nd row	3rd row	Comb.		
	Inner	outer	outer	outer	outer	Inner	Outer
	hair	hair	hair	hair	hair	pillar	pillar
	cells	cells	cells	cells	cells	cells	cells
Group means							
0.125 kHz	0.43	3.01	9.33	14.46	8.93	0.00	0.00
0.25 kHz	0.25	1.33	1.63	4.99	2.65	0.00	0.05
0.5 kHz	0.30	4.44	3.55	3.49	3.82	0.03	0.09
1 kHz	0.25	4.09	5.64	2.42	4.05	0.00	0.00
2 kHz	0.15	5.40	3.12	1.73	3.42	0.00	0.04
4 kHz	0.41	4.09	1.35	1.76	2.40	0.00	0.00
8 kHz	0.25	4.07	1.57	1.28	2.31	0.00	0.00
16 kHz	0.20	2.62	1.18	0.96	1.59	0.00	0.00

Group standard deviations

0.125 kHz	0.71	2.76	5.15	8.73	4.04	0.00	0.00
0.25 kHz	0.48	2.02	1.68	2.96	1.96	0.00	0.14
0.5 kHz	0.41	10.11	6.27	2.83	5.47	0.07	0.25
1 kHz	0.37	9.27	12.41	1.96	7.63	0.00	0.00
2 kHz	0.30	8.88	5.69	1.21	4.86	0.00	0.11
4 kHz	0.49	6.82	2.19	2.08	3.60	0.00	0.00
8 kHz	0.30	9.21	2.82	1.37	4.23	0.00	0.00
16 kHz	0.21	5.07	1.50	0.83	2.27	0.00	0.00

Group standard errors

0.125 kHz	0.25	0.98	1.82	3.09	1.43	0.00	0.00
0.25 kHz	0.17	0.71	0.60	1.05	0.69	0.00	0.05
0.5 kHz	0.15	3.57	2.22	1.00	1.93	0.03	0.09
1 kHz	0.13	3.28	4.39	0.69	2.70	0.00	0.00
2 kHz	0.11	3.14	2.01	0.43	1.72	0.00	0.04
4 kHz	0.17	2.41	0.78	0.74	1.27	0.00	0.00
8 kHz	0.11	3.26	1.00	0.48	1.50	0.00	0.00
16 kHz	0.08	1.79	0.53	0.29	0.80	0.00	0.00

Control Group  
No Noise Exposure

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla A41							
0.125 kHz	3	16	20	34	70	0	0
0.25 kHz	2	2	12	14	28	0	0
0.5 kHz	1	1	6	31	38	0	0
1 kHz	2	10	4	17	31	0	0
2 kHz	0	51	7	4	62	0	0
4 kHz	3	25	3	4	32	0	0
8 kHz	1	89	28	9	126	0	0
16 kHz	1	44	13	6	63	0	0
TOTALS	13	238	93	119	450	0	0
Chinchilla A52							
0.125 kHz	0	10	23	25	58	0	0
0.25 kHz	0	3	2	15	20	0	1
0.5 kHz	0	5	5	5	15	0	2
1 kHz	0	2	0	4	6	0	0
2 kHz	0	2	6	10	18	0	0
4 kHz	2	1	5	6	12	0	0
8 kHz	0	3	5	4	12	0	0
16 kHz	0	6	0	0	6	0	0
TOTALS	2	32	46	69	147	0	3
Chinchilla A61							
0.125 kHz	0	4	14	22	40	0	0
0.25 kHz	3	20	16	32	68	0	0
0.5 kHz	3	96	62	10	168	0	0
1 kHz	2	83	112	16	211	0	0
2 kHz	0	74	54	10	138	0	1
4 kHz	0	63	21	21	105	0	0
8 kHz	2	7	3	12	22	0	0
16 kHz	1	1	1	6	8	0	0
TOTALS	11	348	283	129	760	0	1

Control Group  
No Noise Exposure

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla A89							
0.125 kHz	0	4	4	38	46	0	0
0.25 kHz	0	1	2	6	9	0	0
0.5 kHz	1	3	6	4	13	1	0
1 kHz	0	3	13	8	24	0	0
2 kHz	0	5	8	2	15	0	0
4 kHz	0	4	1	2	7	0	0
8 kHz	1	0	0	1	1	0	0
16 kHz	1	1	0	0	1	0	0
TOTALS	3	21	34	61	116	1	0
Chinchilla B17							
0.125 kHz	1	3	9	9	21	0	0
0.25 kHz	0	1	4	17	22	0	0
0.5 kHz	1	1	4	18	23	0	0
1 kHz	0	0	2	10	12	0	0
2 kHz	2	2	2	1	5	0	0
4 kHz	0	5	2	2	9	0	0
8 kHz	0	3	3	2	8	0	0
16 kHz	0	0	5	4	9	0	0
TOTALS	4	15	31	63	109	0	0
Chinchilla B86							
0.125 kHz	1	0	10	20	30	0	0
0.25 kHz	0	1	1	2	4	0	0
0.5 kHz	0	1	1	2	4	0	0
1 kHz	0	0	2	1	3	0	0
2 kHz	0	1	1	4	6	0	0
4 kHz	0	2	1	0	3	0	0
8 kHz	1	3	1	0	4	0	0
16 kHz	1	1	0	1	2	0	0
TOTALS	3	9	17	30	56	0	0

Control Group  
No Noise Exposure

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla B96							
0.125 kHz	0	2	30	8	40	0	0
0.25 kHz	0	0	1	25	26	0	0
0.5 kHz	0	2	3	16	21	0	0
1 kHz	0	0	3	1	4	0	0
2 kHz	0	1	1	7	9	0	0
4 kHz	1	3	0	6	9	0	0
8 kHz	0	2	1	0	3	0	0
16 kHz	0	0	5	3	8	0	0
TOTALS	1	10	44	66	120	0	0
Chinchilla Z87							
0.125 kHz	0	5	26	57	88	0	0
0.25 kHz	0	6	5	18	29	0	0
0.5 kHz	0	6	5	6	17	0	0
1 kHz	1	3	4	4	11	0	0
2 kHz	1	3	0	4	7	0	0
4 kHz	2	2	1	3	6	0	0
8 kHz	0	0	0	4	4	0	0
16 kHz	0	7	3	2	12	0	0
TOTALS	4	32	44	98	174	0	0

Control Group  
No Noise Exposure

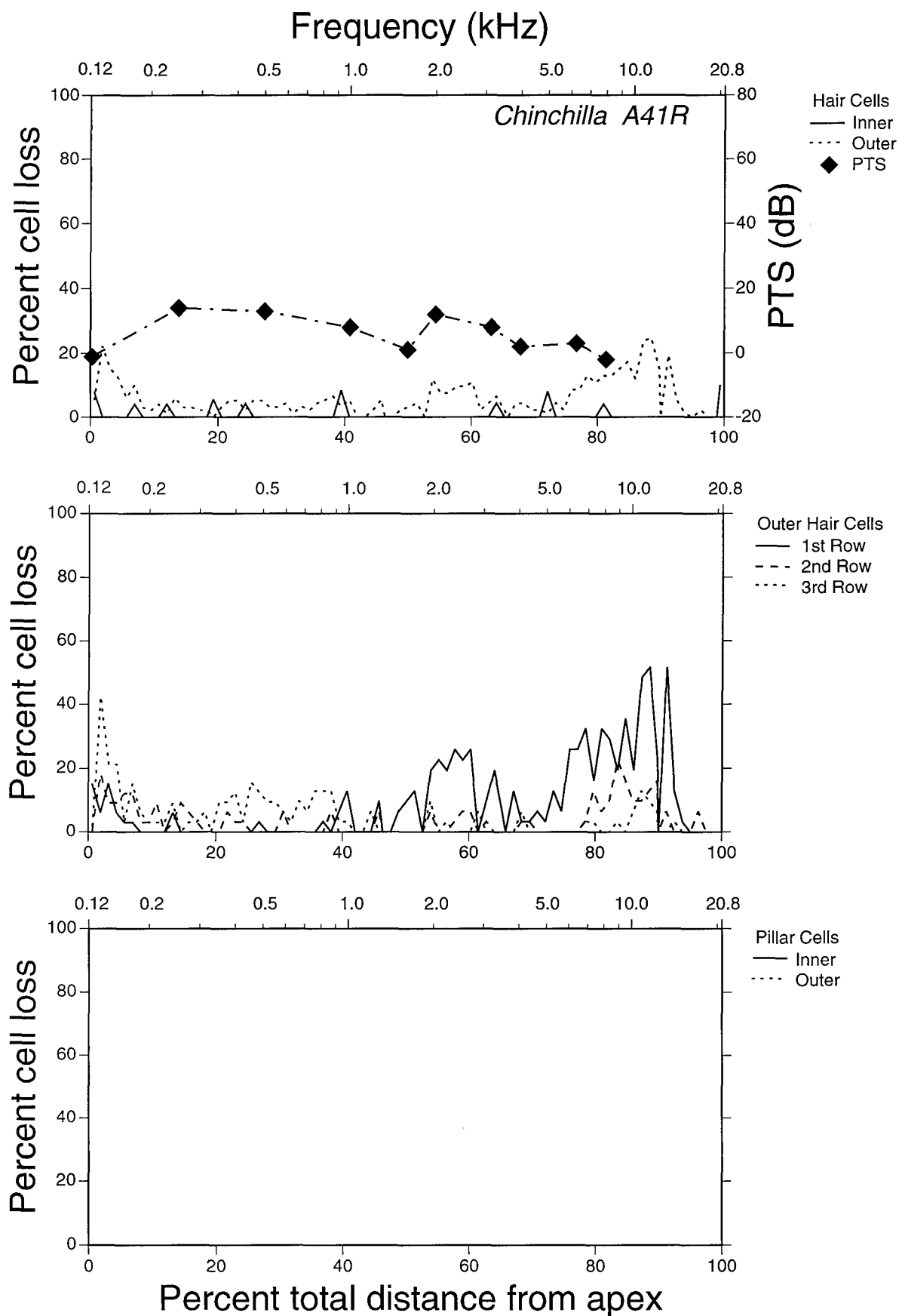
Percent sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla A41							
0.125 kHz	2.0	8.2	10.3	17.4	12.0	0.0	0.0
0.25 kHz	0.8	0.6	3.5	4.1	2.7	0.0	0.0
0.5 kHz	0.4	0.3	1.8	9.1	3.7	0.0	0.0
1 kHz	0.8	3.1	1.2	5.2	3.2	0.0	0.0
2 kHz	0.0	15.4	2.1	1.2	6.2	0.0	0.0
4 kHz	1.2	7.6	0.9	1.2	3.2	0.0	0.0
8 kHz	0.4	26.8	8.4	2.7	12.6	0.0	0.0
16 kHz	0.4	14.9	4.4	2.0	7.1	0.0	0.0
Chinchilla A52							
0.125 kHz	0.0	6.2	14.3	15.5	12.0	0.0	0.0
0.25 kHz	0.0	1.1	0.7	5.3	2.4	0.0	0.4
0.5 kHz	0.0	1.8	1.8	1.8	1.8	0.0	0.7
1 kHz	0.0	0.7	0.0	1.5	0.7	0.0	0.0
2 kHz	0.0	0.7	2.2	3.7	2.2	0.0	0.0
4 kHz	0.9	0.4	1.8	2.2	1.5	0.0	0.0
8 kHz	0.0	1.1	1.8	1.5	1.5	0.0	0.0
16 kHz	0.0	2.5	0.0	0.0	0.8	0.0	0.0
Chinchilla A61							
0.125 kHz	0.0	2.2	7.5	11.8	7.2	0.0	0.0
0.25 kHz	1.2	6.1	4.9	9.8	6.9	0.0	0.0
0.5 kHz	1.2	29.4	19.0	3.1	17.2	0.0	0.0
1 kHz	0.8	26.9	36.2	5.2	22.8	0.0	0.0
2 kHz	0.0	23.3	17.0	3.2	14.5	0.0	0.3
4 kHz	0.0	19.9	6.6	6.6	11.0	0.0	0.0
8 kHz	0.8	2.2	0.9	3.8	2.3	0.0	0.0
16 kHz	0.4	0.4	0.4	2.1	1.0	0.0	0.0
Chinchilla A89							
0.125 kHz	0.0	2.1	2.1	20.1	8.1	0.0	0.0
0.25 kHz	0.0	0.3	0.6	1.8	0.9	0.0	0.0
0.5 kHz	0.4	0.9	1.8	1.2	1.3	0.2	0.0
1 kHz	0.0	1.0	4.1	2.5	2.5	0.0	0.0
2 kHz	0.0	1.6	2.5	0.6	1.6	0.0	0.0
4 kHz	0.0	1.2	0.3	0.6	0.7	0.0	0.0
8 kHz	0.4	0.0	0.0	0.3	0.1	0.0	0.0
16 kHz	0.4	0.3	0.0	0.0	0.1	0.0	0.0

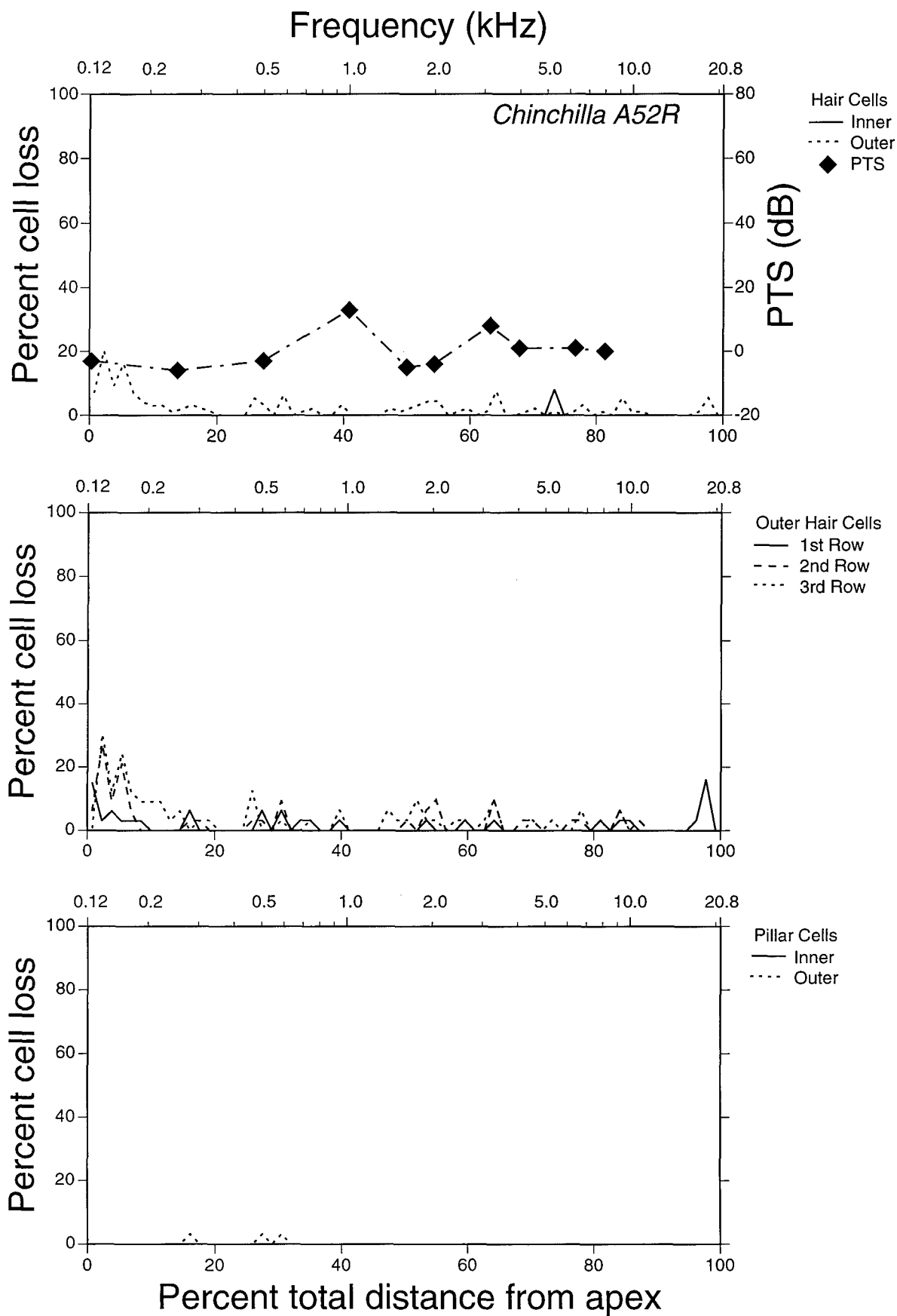
Control Group  
No Noise Exposure

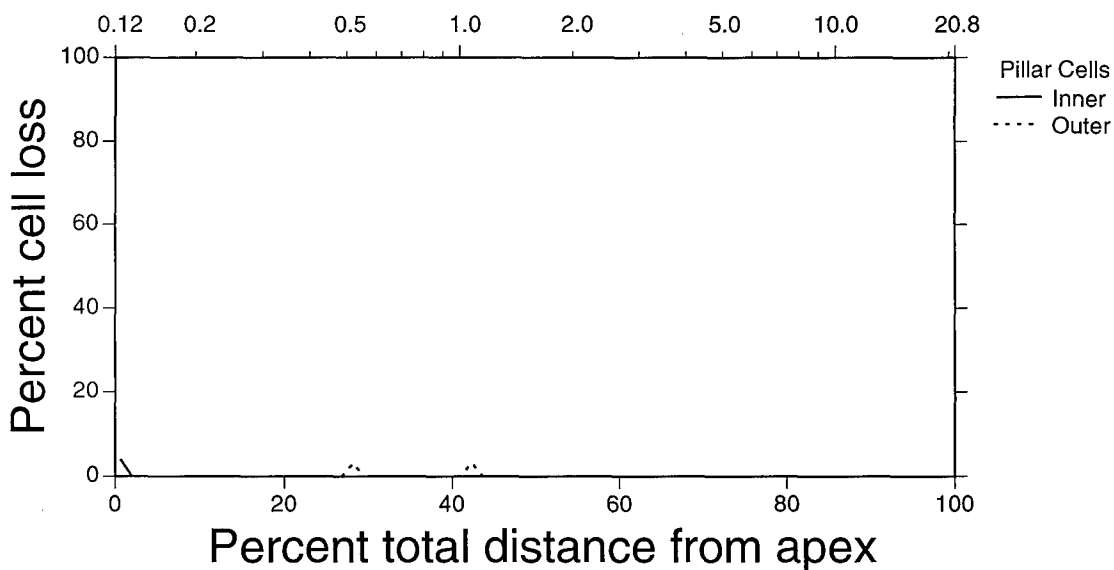
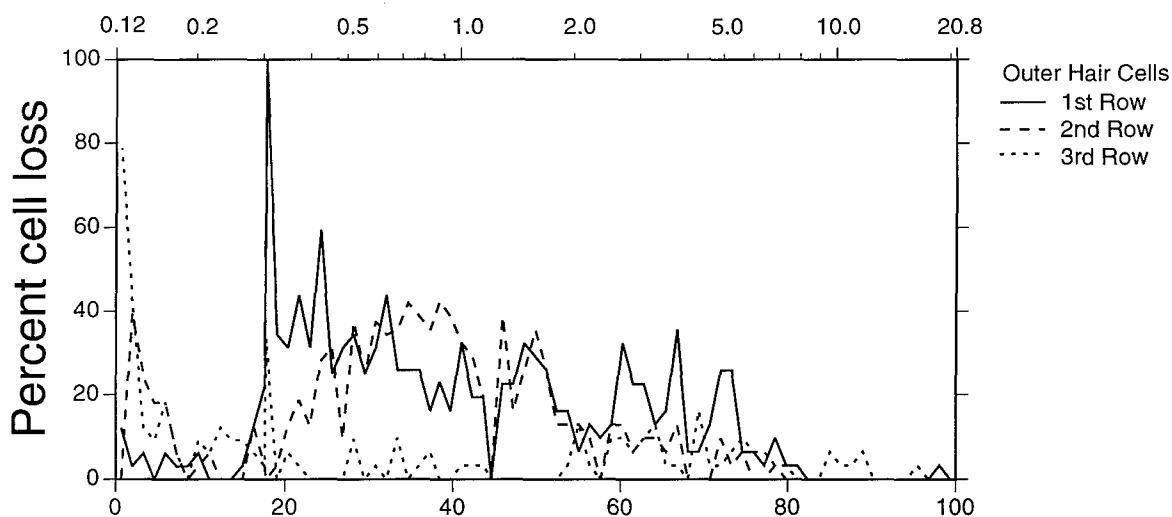
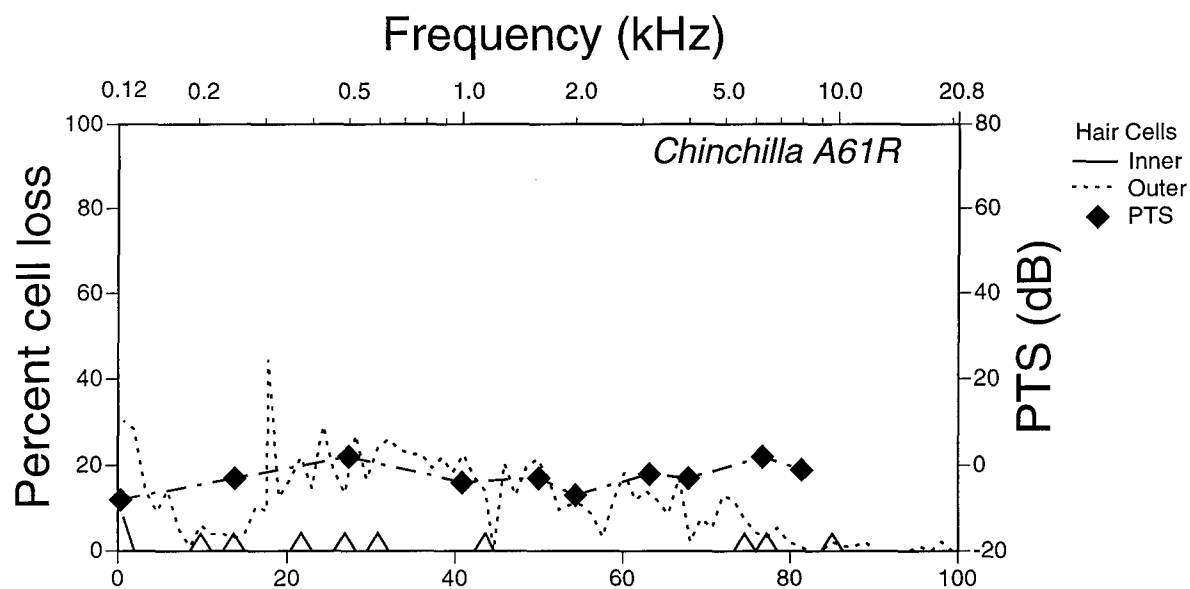
Percent sensory cell losses over octave band frequencies

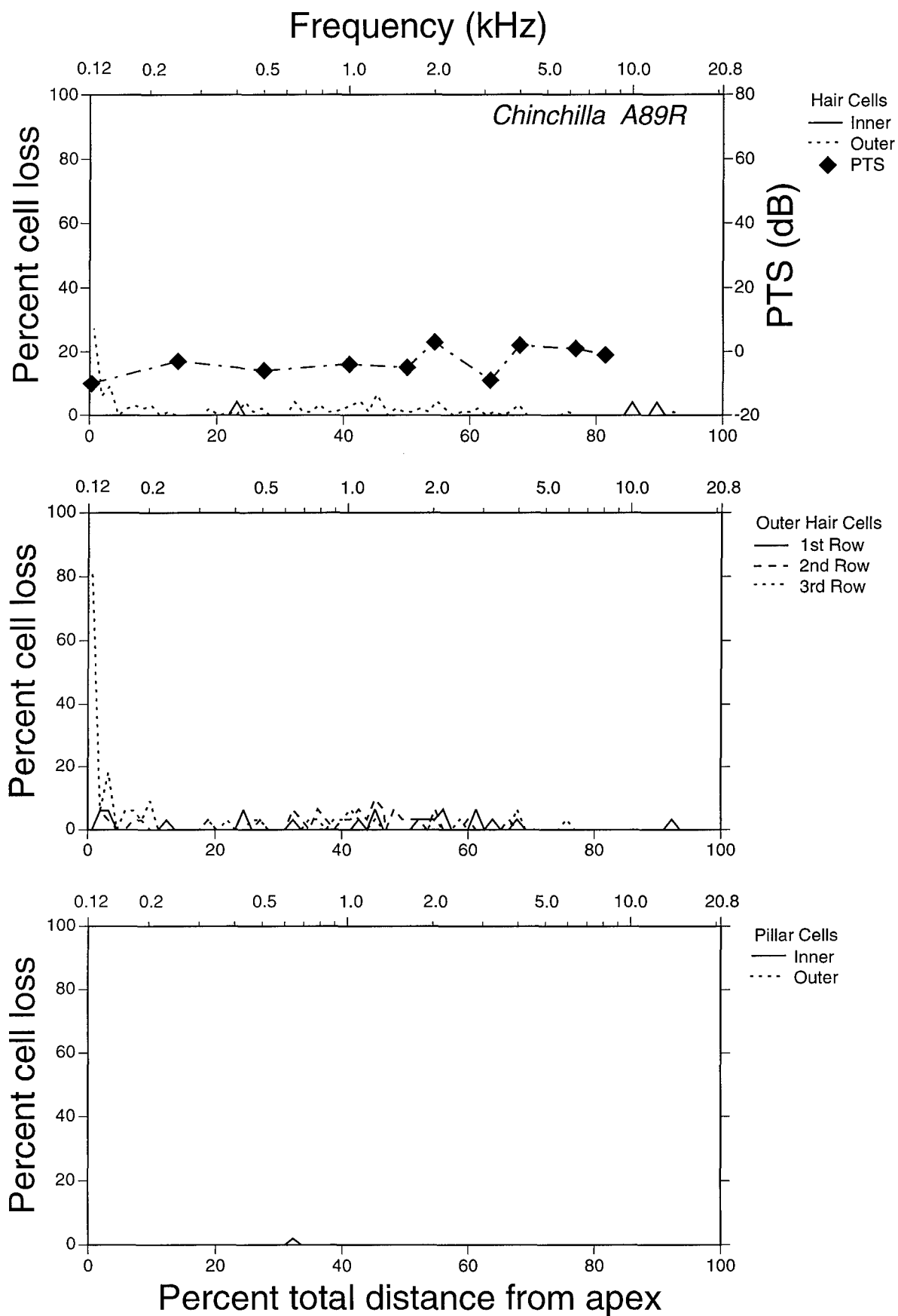
	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla B17							
0.125 kHz	0.7	1.6	4.7	4.7	3.7	0.0	0.0
0.25 kHz	0.0	0.3	1.2	5.0	2.2	0.0	0.0
0.5 kHz	0.4	0.3	1.2	5.3	2.3	0.0	0.0
1 kHz	0.0	0.0	0.6	3.1	1.2	0.0	0.0
2 kHz	0.8	0.6	0.6	0.3	0.5	0.0	0.0
4 kHz	0.0	1.5	0.6	0.6	0.9	0.0	0.0
8 kHz	0.0	0.9	0.9	0.6	0.8	0.0	0.0
16 kHz	0.0	0.0	1.7	1.4	1.0	0.0	0.0
Chinchilla B86							
0.125 kHz	0.7	0.0	5.5	10.9	5.5	0.0	0.0
0.25 kHz	0.0	0.3	0.3	0.6	0.4	0.0	0.0
0.5 kHz	0.0	0.3	0.3	0.6	0.4	0.0	0.0
1 kHz	0.0	0.0	0.7	0.3	0.3	0.0	0.0
2 kHz	0.0	0.3	0.3	1.3	0.6	0.0	0.0
4 kHz	0.0	0.6	0.3	0.0	0.3	0.0	0.0
8 kHz	0.4	1.0	0.3	0.0	0.4	0.0	0.0
16 kHz	0.4	0.4	0.0	0.4	0.3	0.0	0.0
Chinchilla B96							
0.125 kHz	0.0	1.1	16.1	4.3	7.2	0.0	0.0
0.25 kHz	0.0	0.0	0.3	7.7	2.7	0.0	0.0
0.5 kHz	0.0	0.6	0.9	4.9	2.1	0.0	0.0
1 kHz	0.0	0.0	1.0	0.3	0.4	0.0	0.0
2 kHz	0.0	0.3	0.3	2.2	0.9	0.0	0.0
4 kHz	0.4	0.9	0.0	1.9	0.9	0.0	0.0
8 kHz	0.0	0.6	0.3	0.0	0.3	0.0	0.0
16 kHz	0.0	0.0	1.8	1.1	1.0	0.0	0.0
Chinchilla Z87							
0.125 kHz	0.0	2.7	14.1	31.0	15.9	0.0	0.0
0.25 kHz	0.0	1.9	1.5	5.6	3.0	0.0	0.0
0.5 kHz	0.0	1.9	1.6	1.9	1.8	0.0	0.0
1 kHz	0.4	1.0	1.3	1.3	1.2	0.0	0.0
2 kHz	0.4	1.0	0.0	1.3	0.8	0.0	0.0
4 kHz	0.8	0.6	0.3	1.0	0.6	0.0	0.0
8 kHz	0.0	0.0	0.0	1.3	0.4	0.0	0.0
16 kHz	0.0	2.5	1.1	0.7	1.4	0.0	0.0

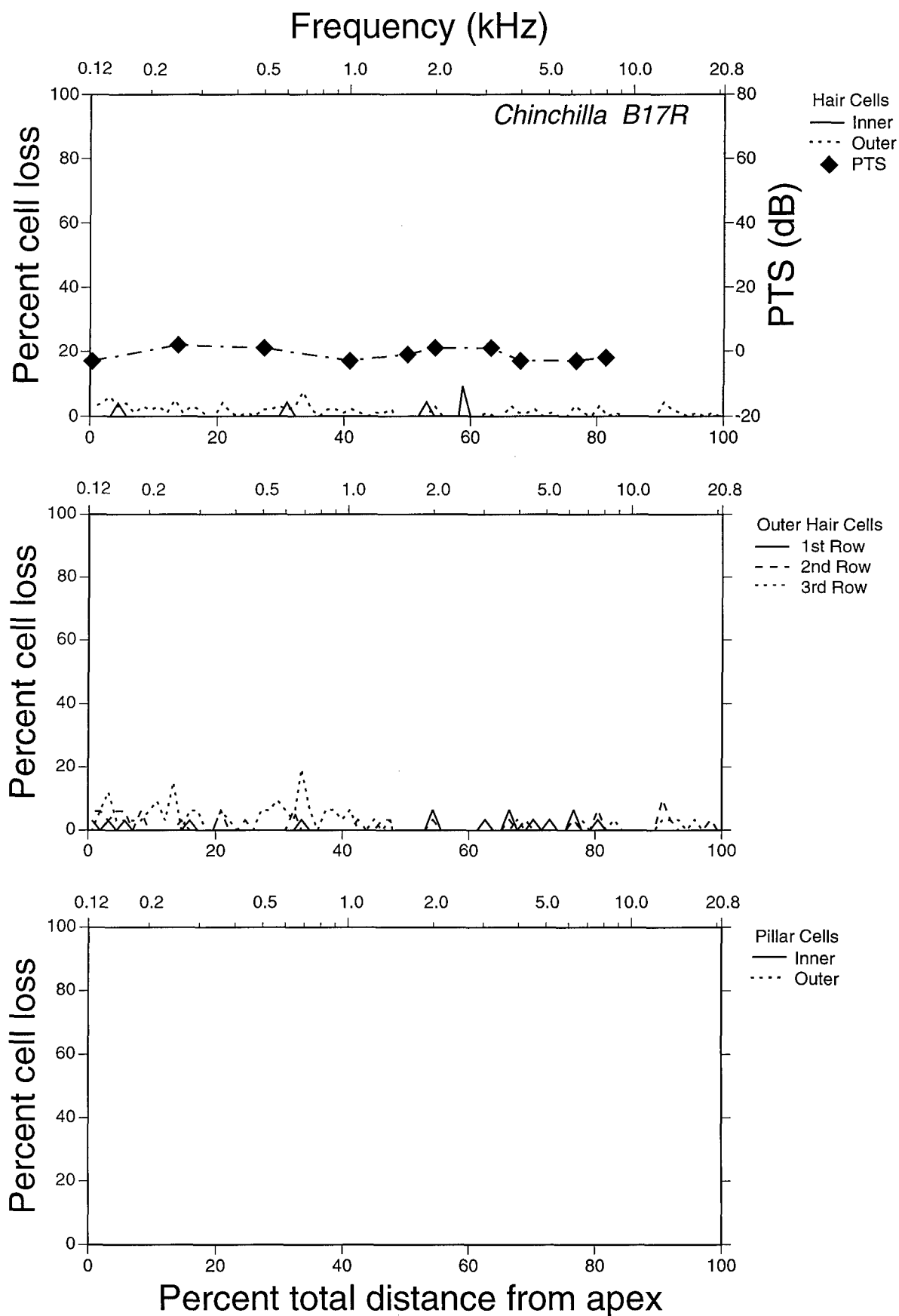


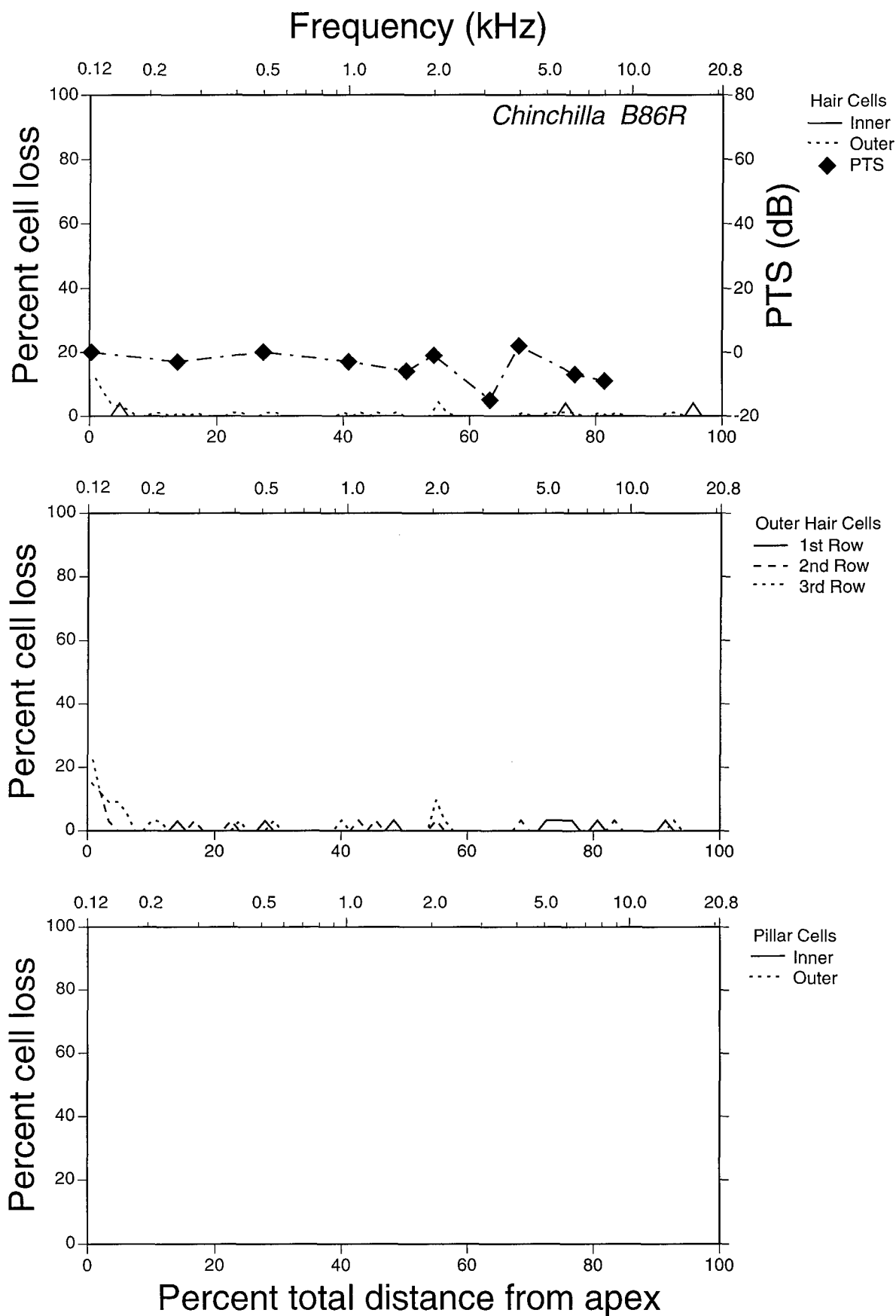


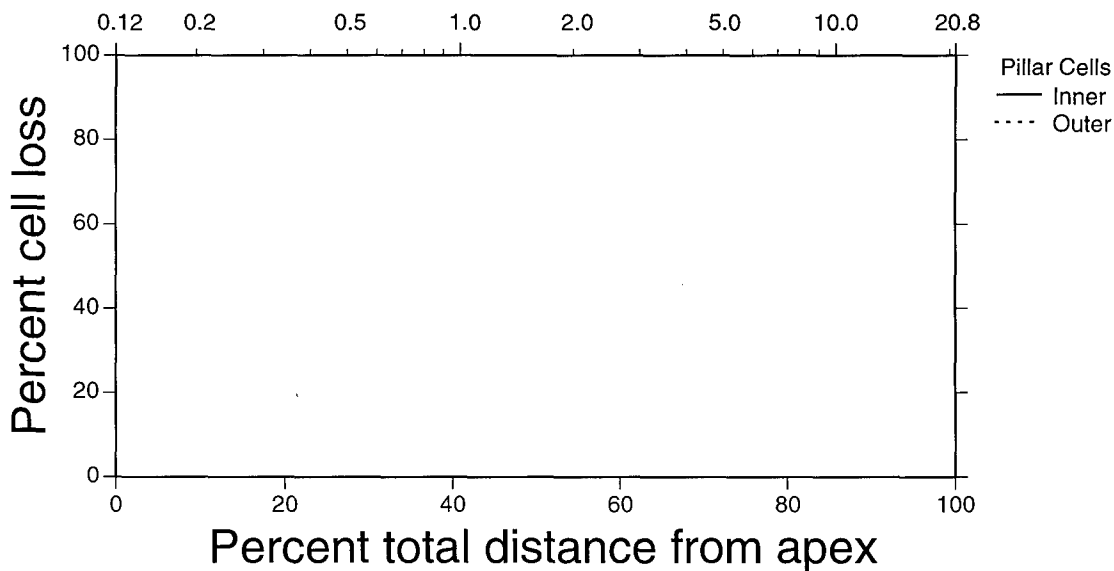
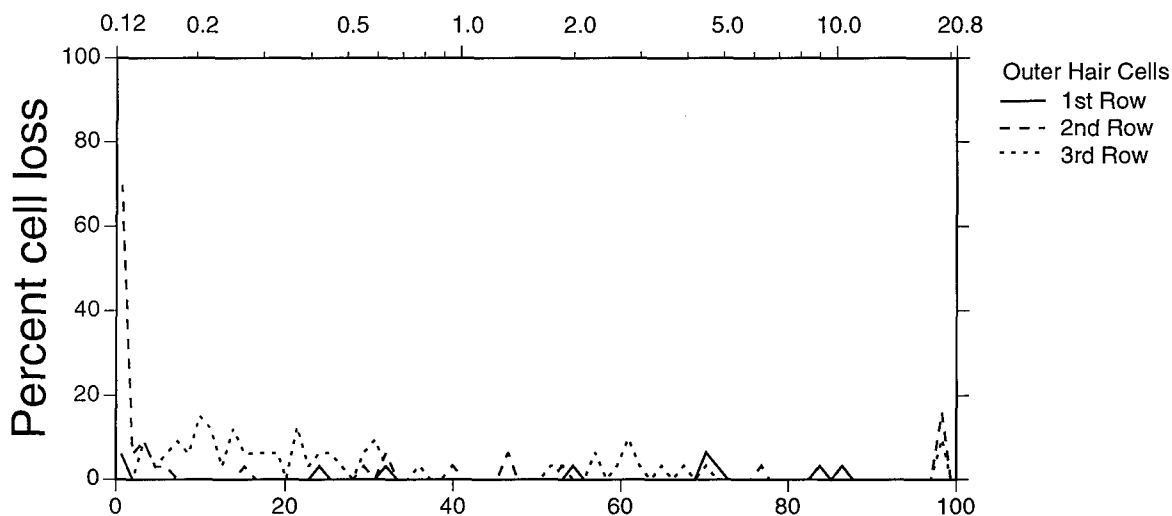
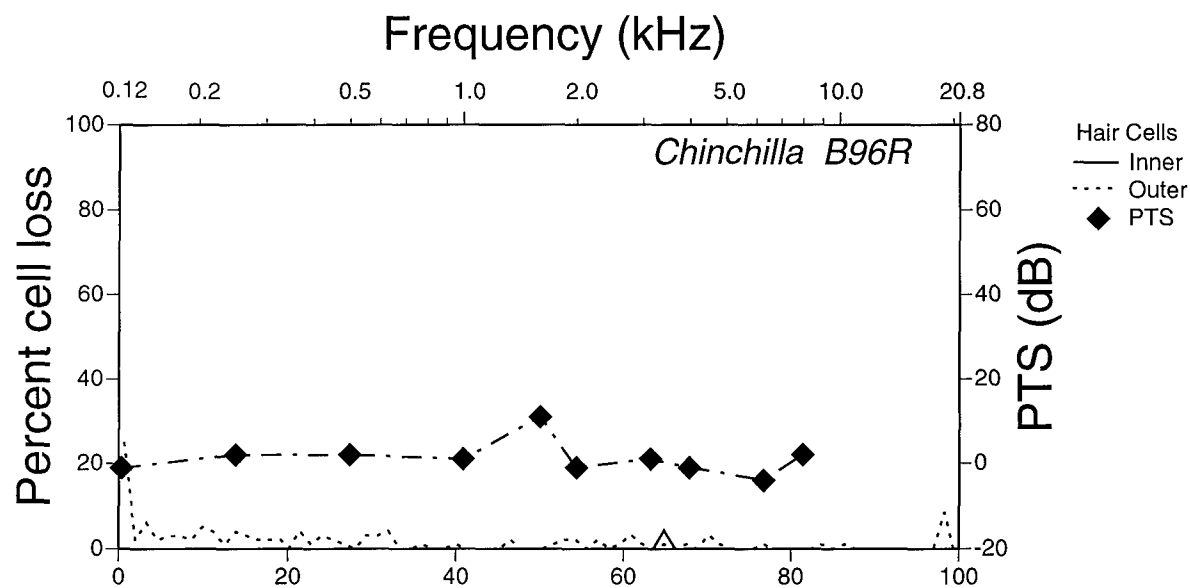


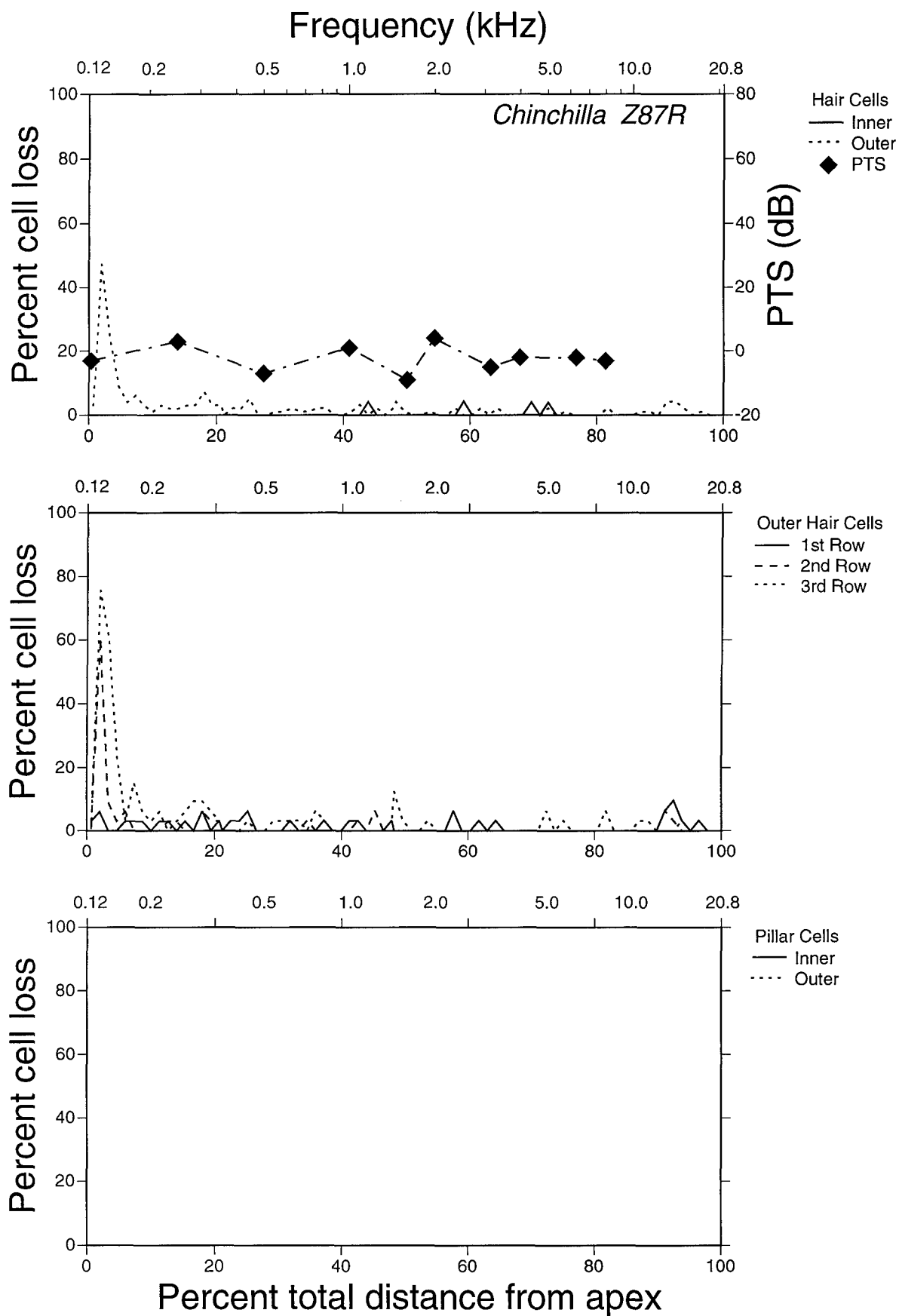








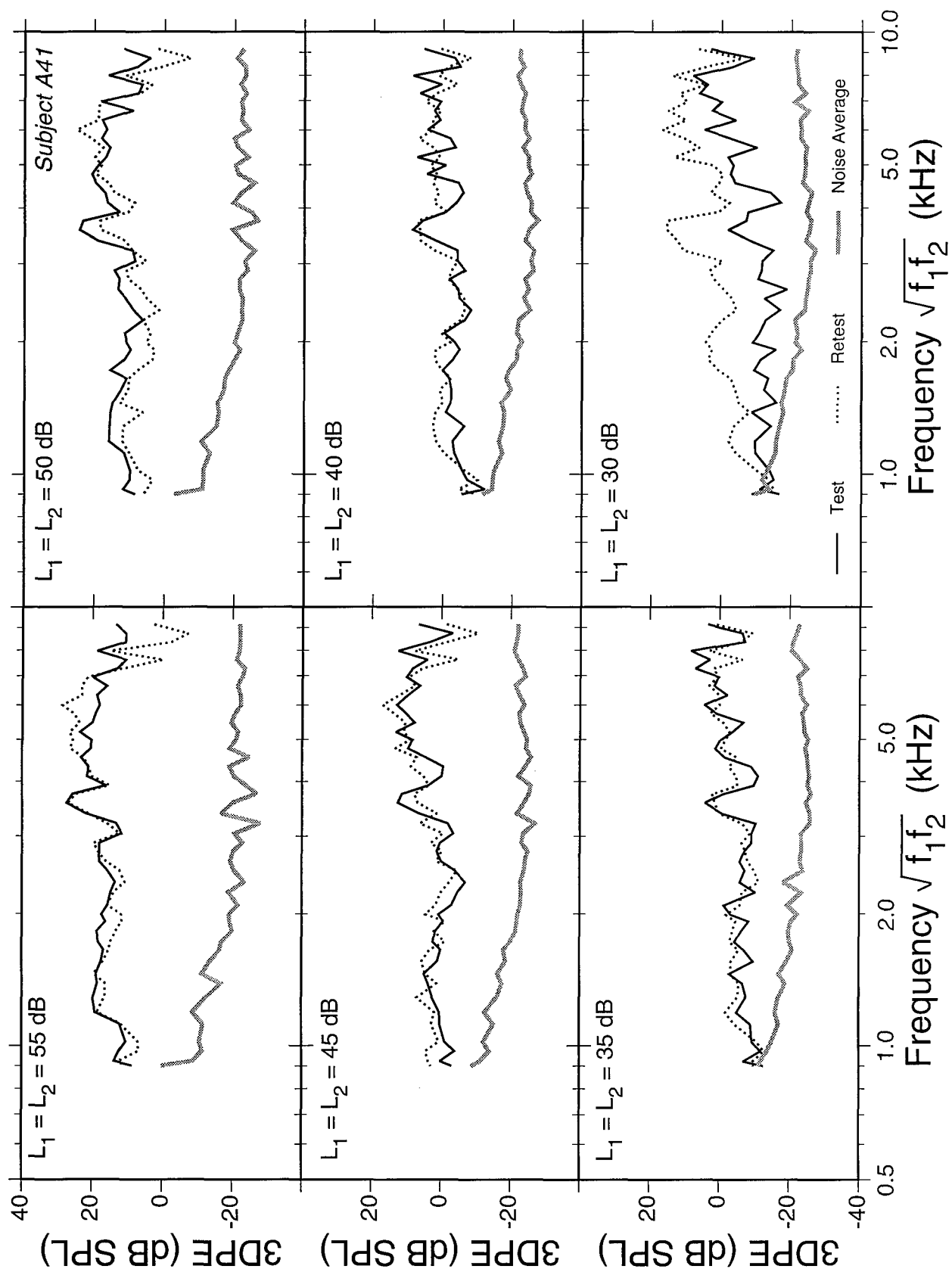


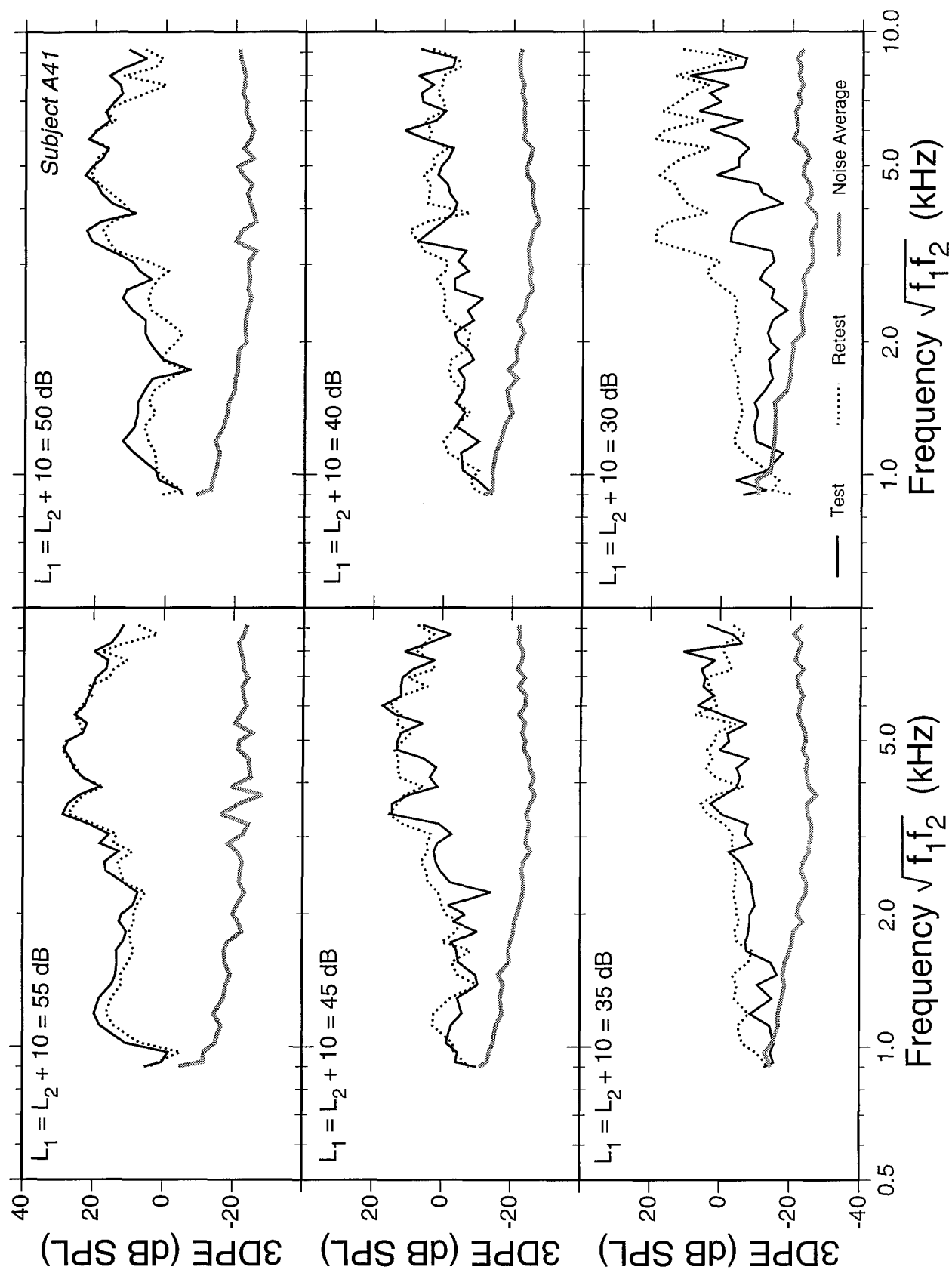


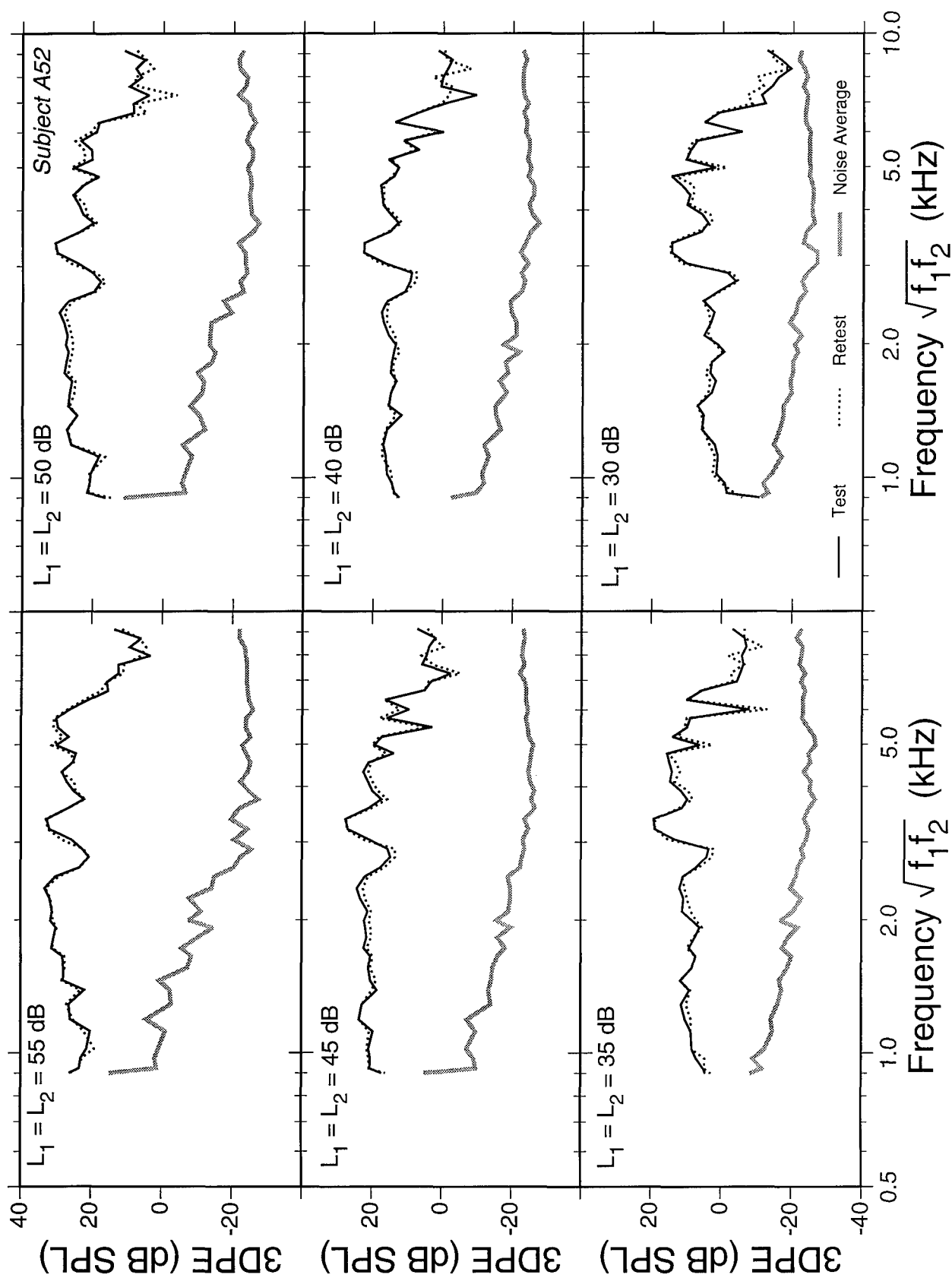
## Individual Control Subject DPEgrams

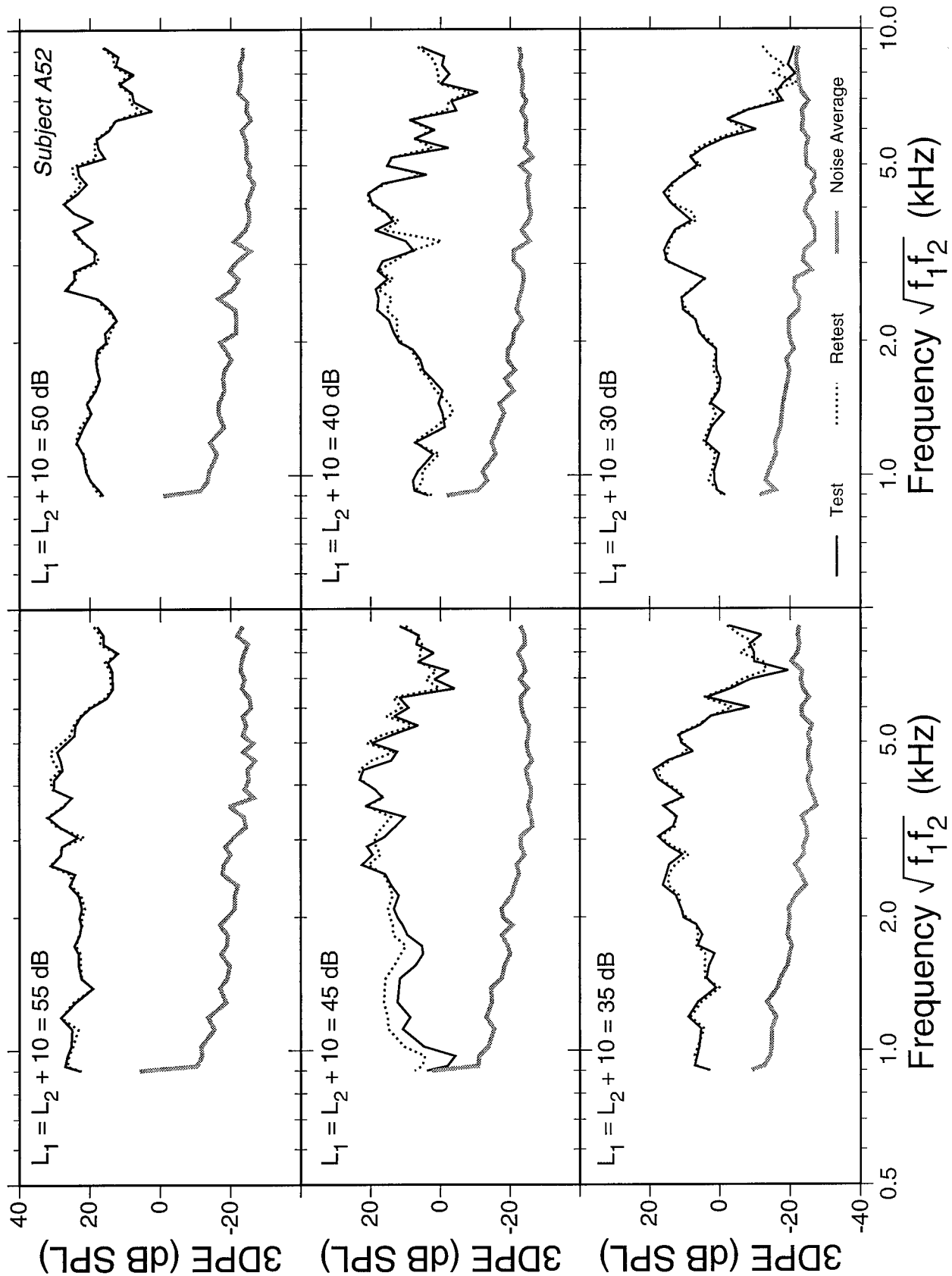
Group mean DPEgrams were presented at the beginning of this data appendix (Pages 154 and 155). The next set of figures (Pages 180 through 197) show the test and retest DPEgrams for the nine individual animals that make up this control group (see Page 151). Animal B84 died during the second set of DPEgram measurements and therefore was not included in the group mean DPEgram graphs. The solid lines represent the first set of DPEgram measurements at the six equal-primary ( $L_1 = L_2 = 55$  to 30 dB SPL) and six unequal-primary ( $L_1 = L_2 + 10 = 55$  to 30 dB SPL) levels. Each subject's DPEgrams represent the average of three measurements made on different days. The dashed lines represent the mean of three DPEgram measurements made on separate days at least 14 days following the first set of measurements. The thick gray line represents the average noise floor over the entire series of emissions measurements.

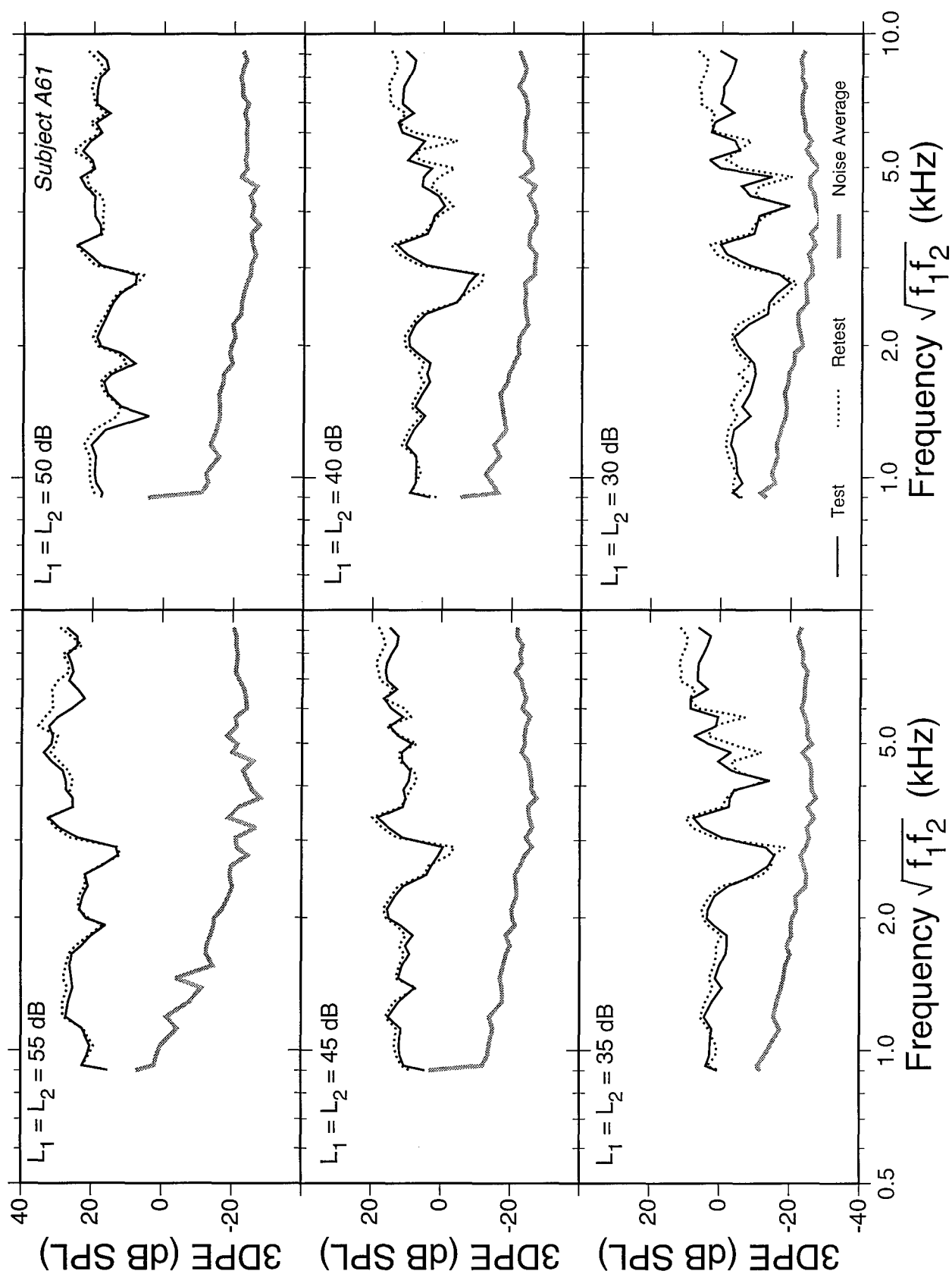


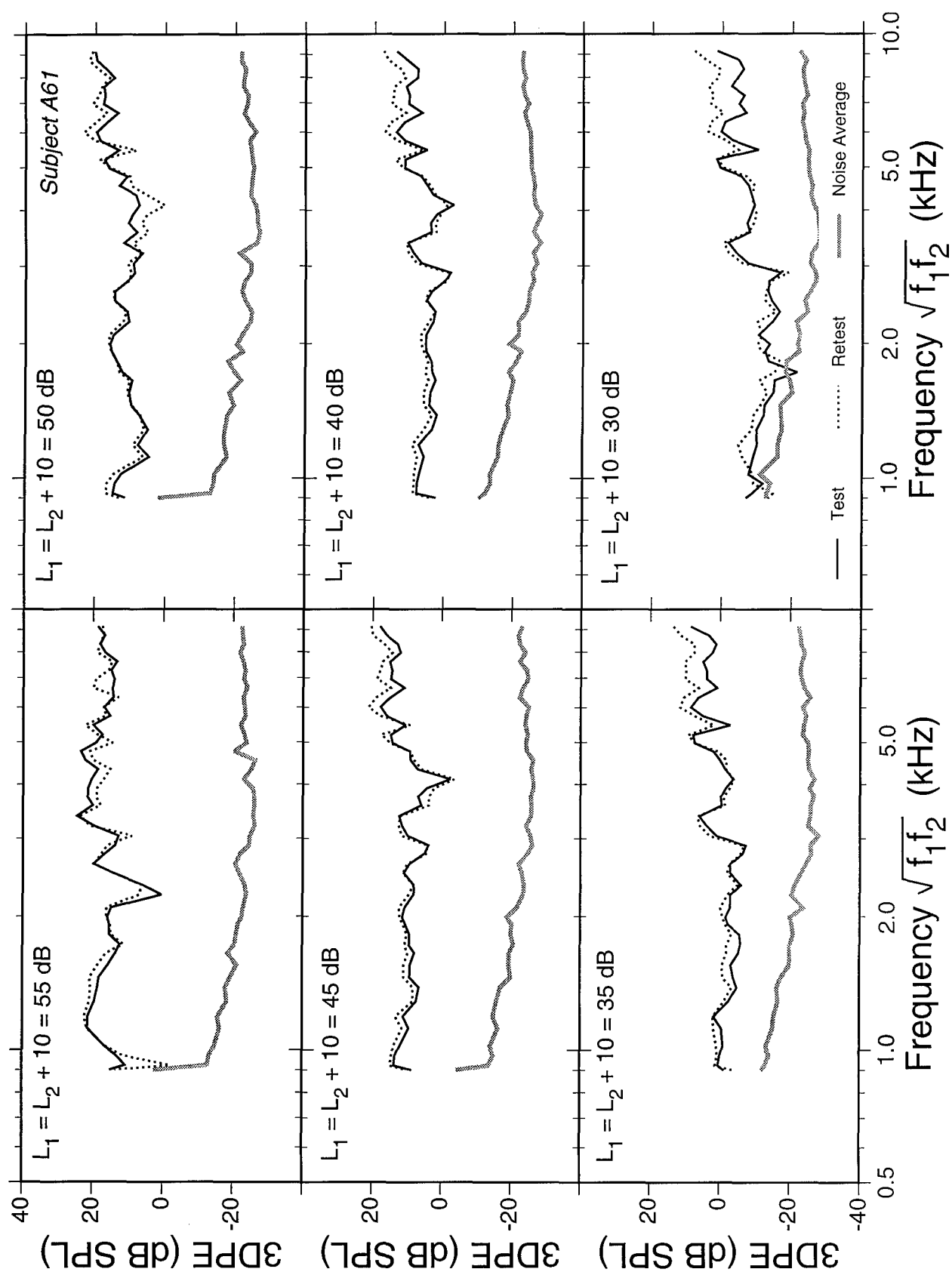


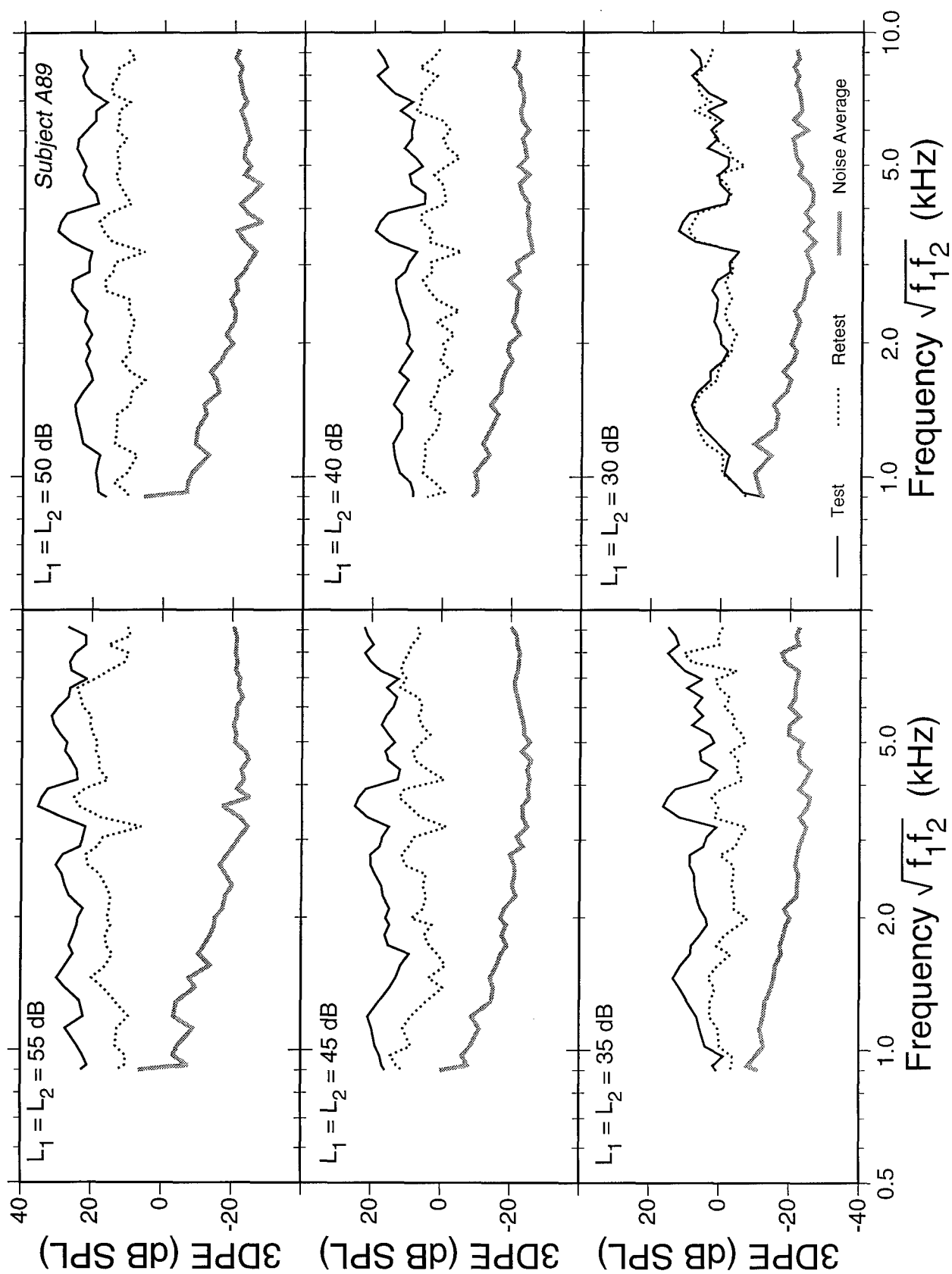


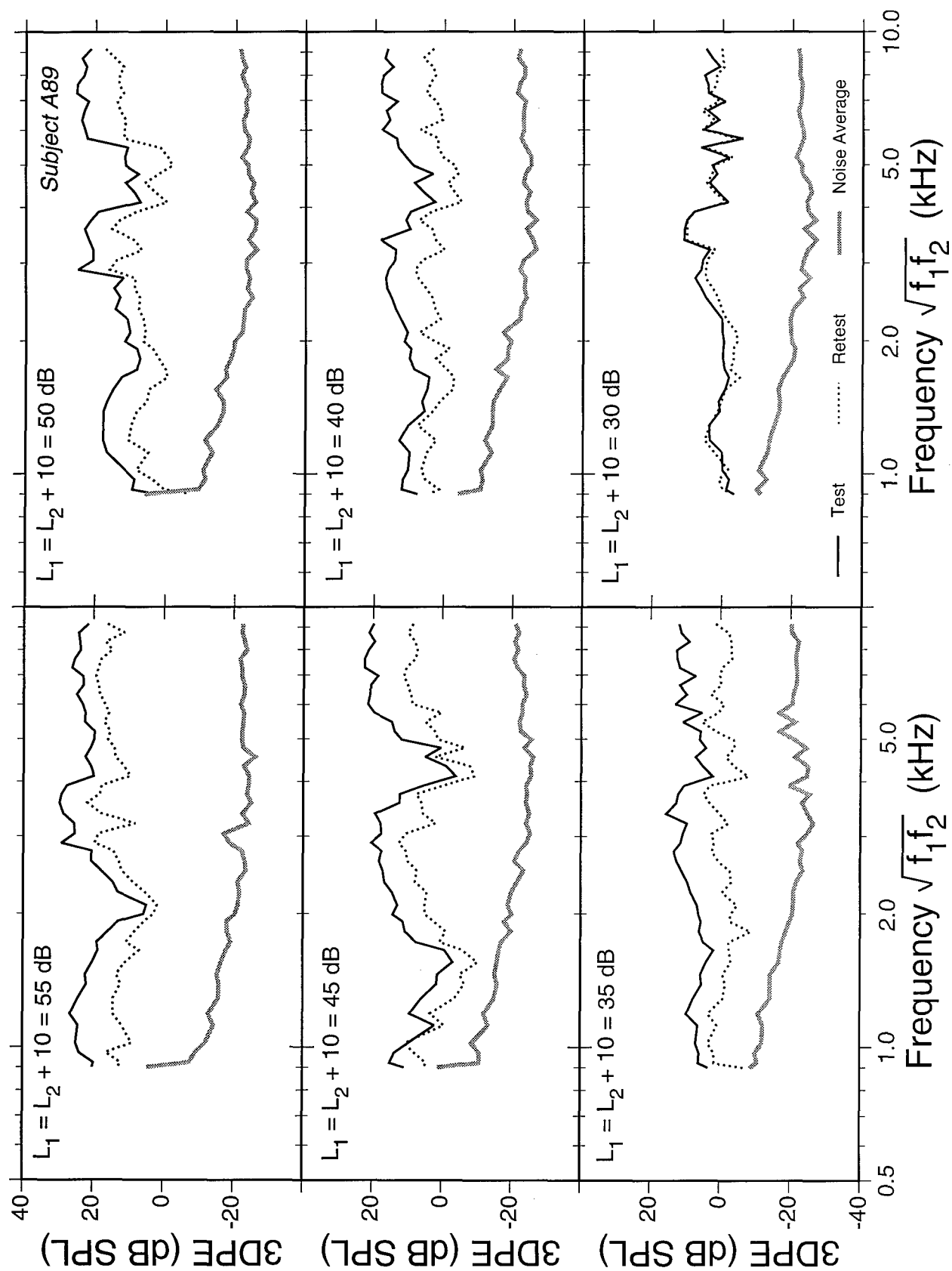




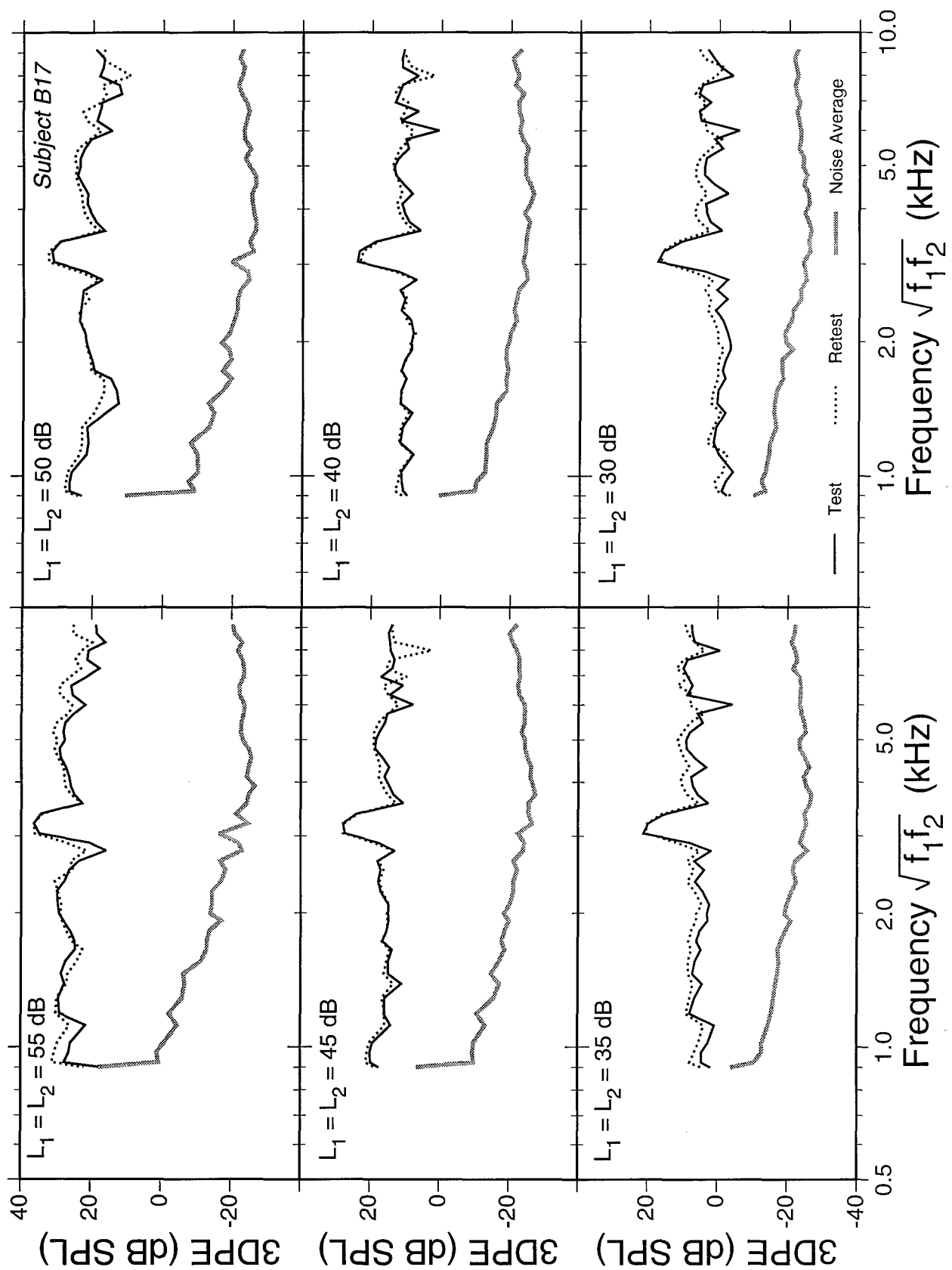


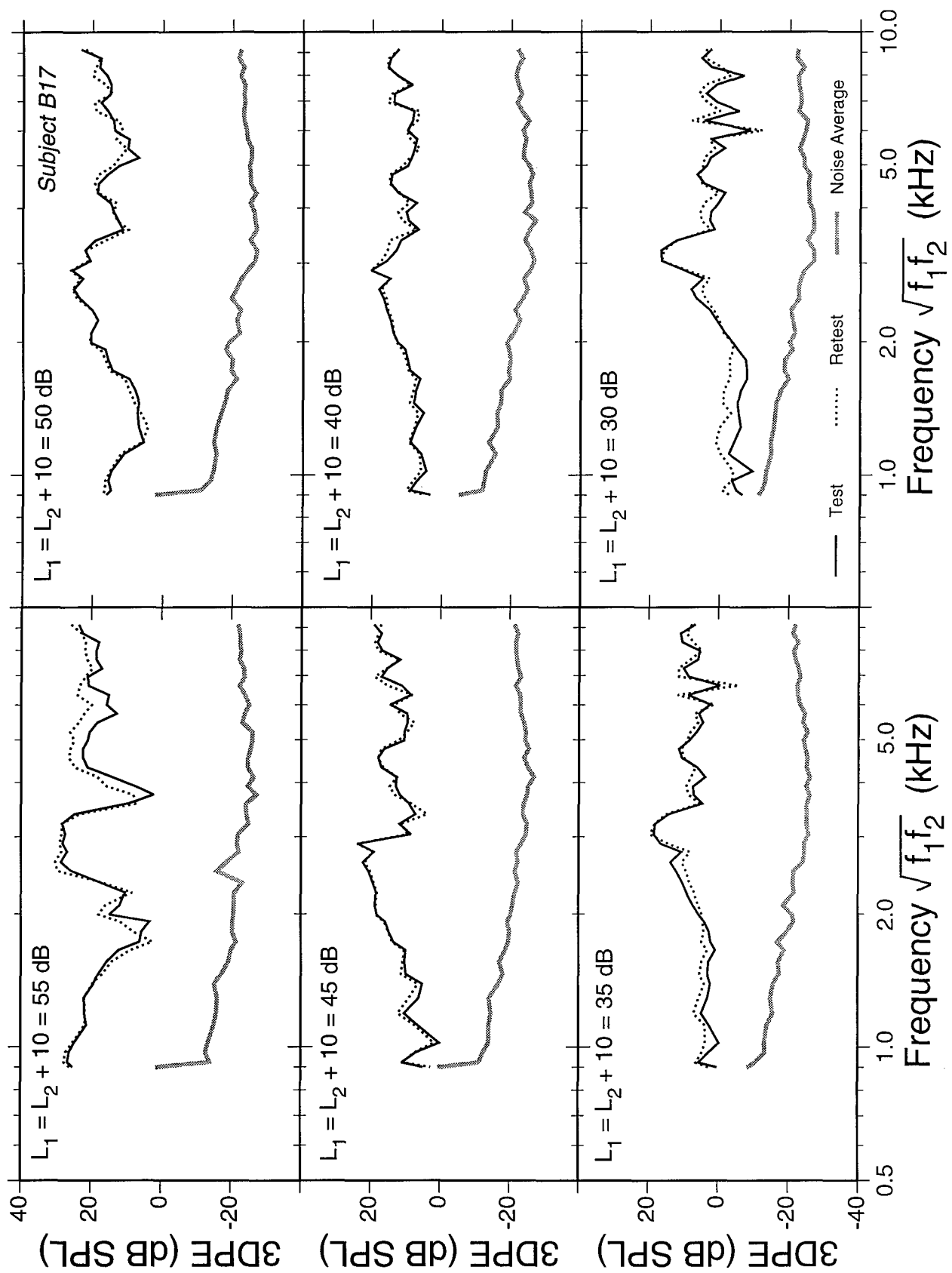


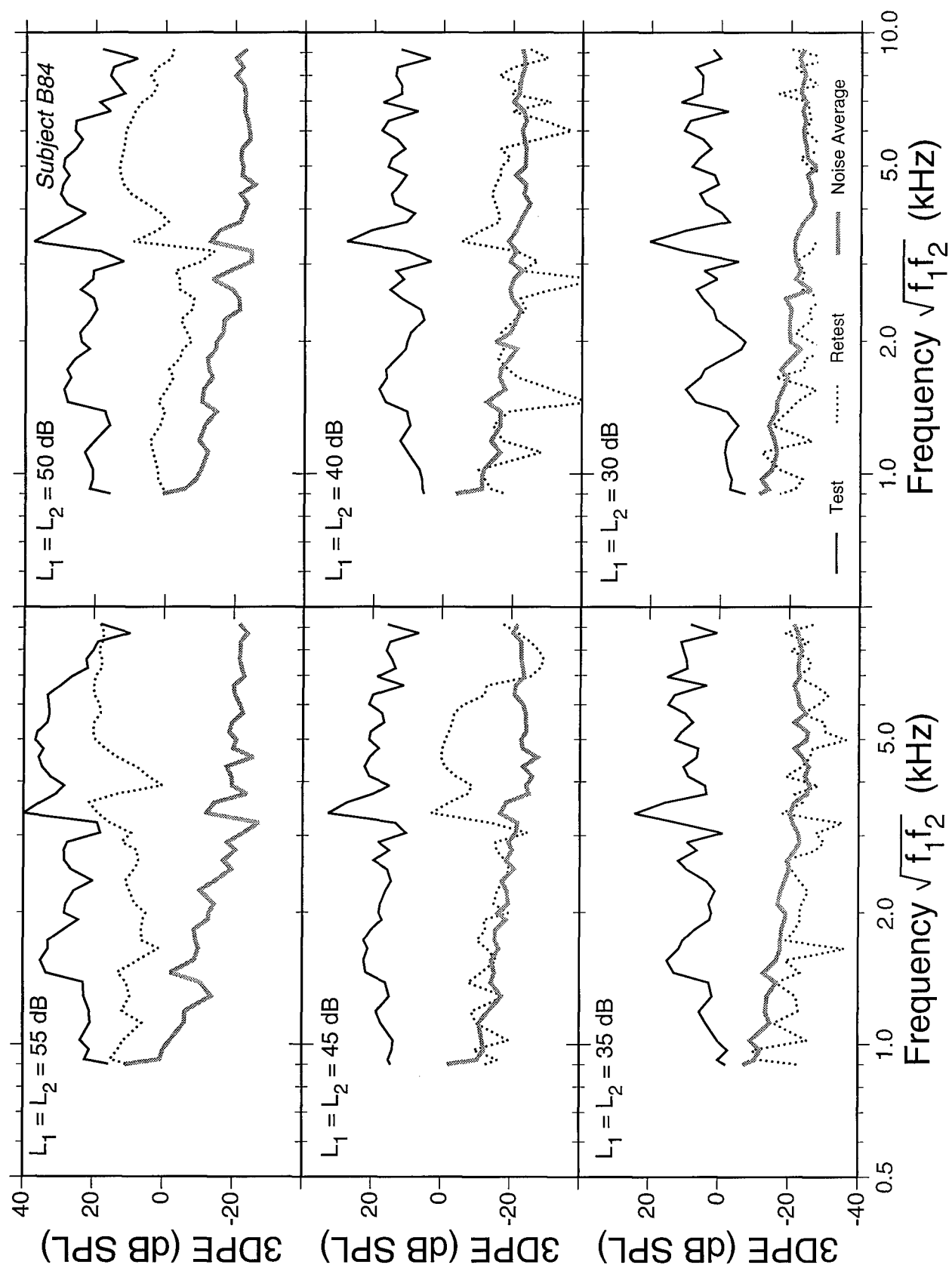


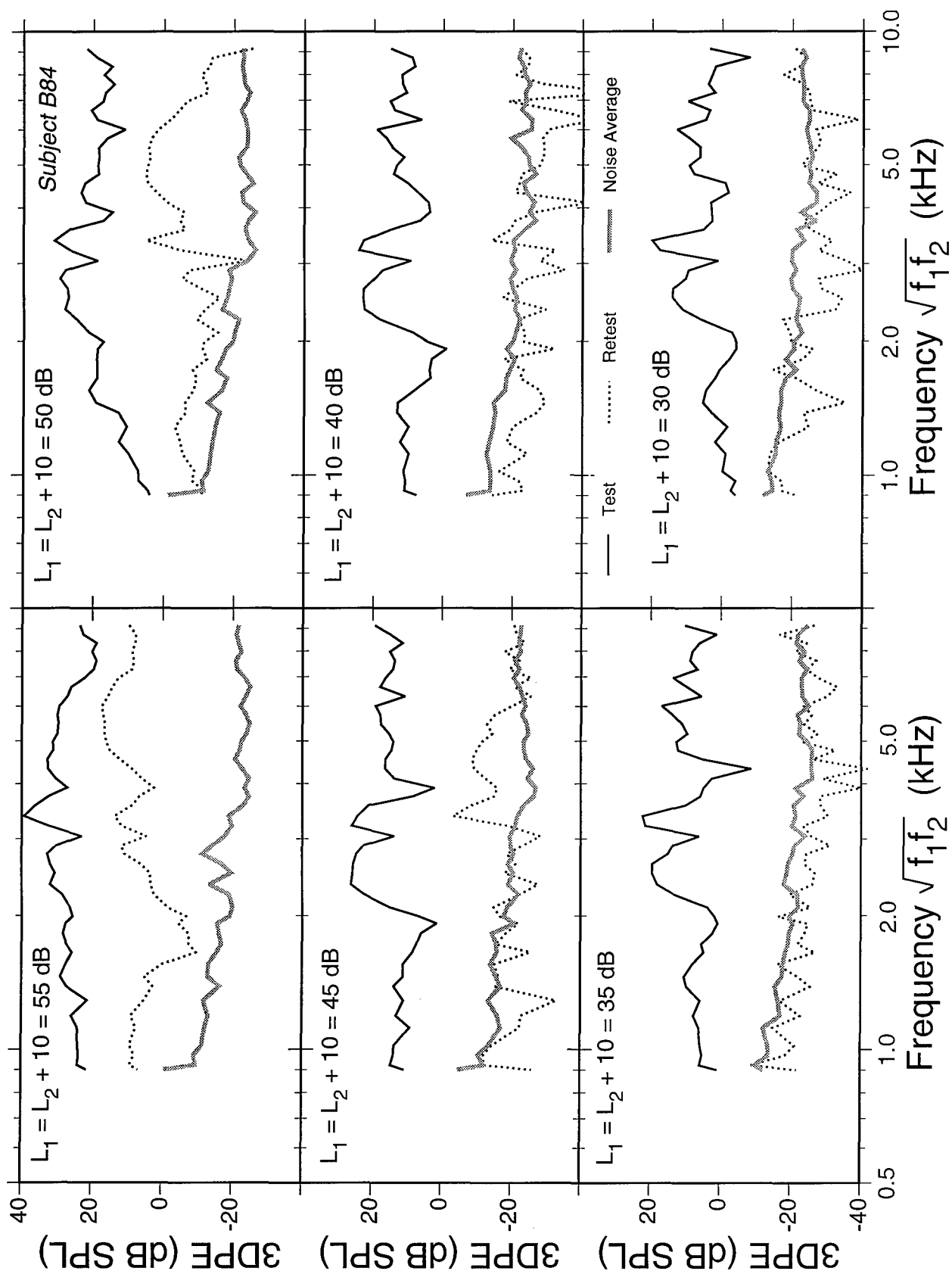


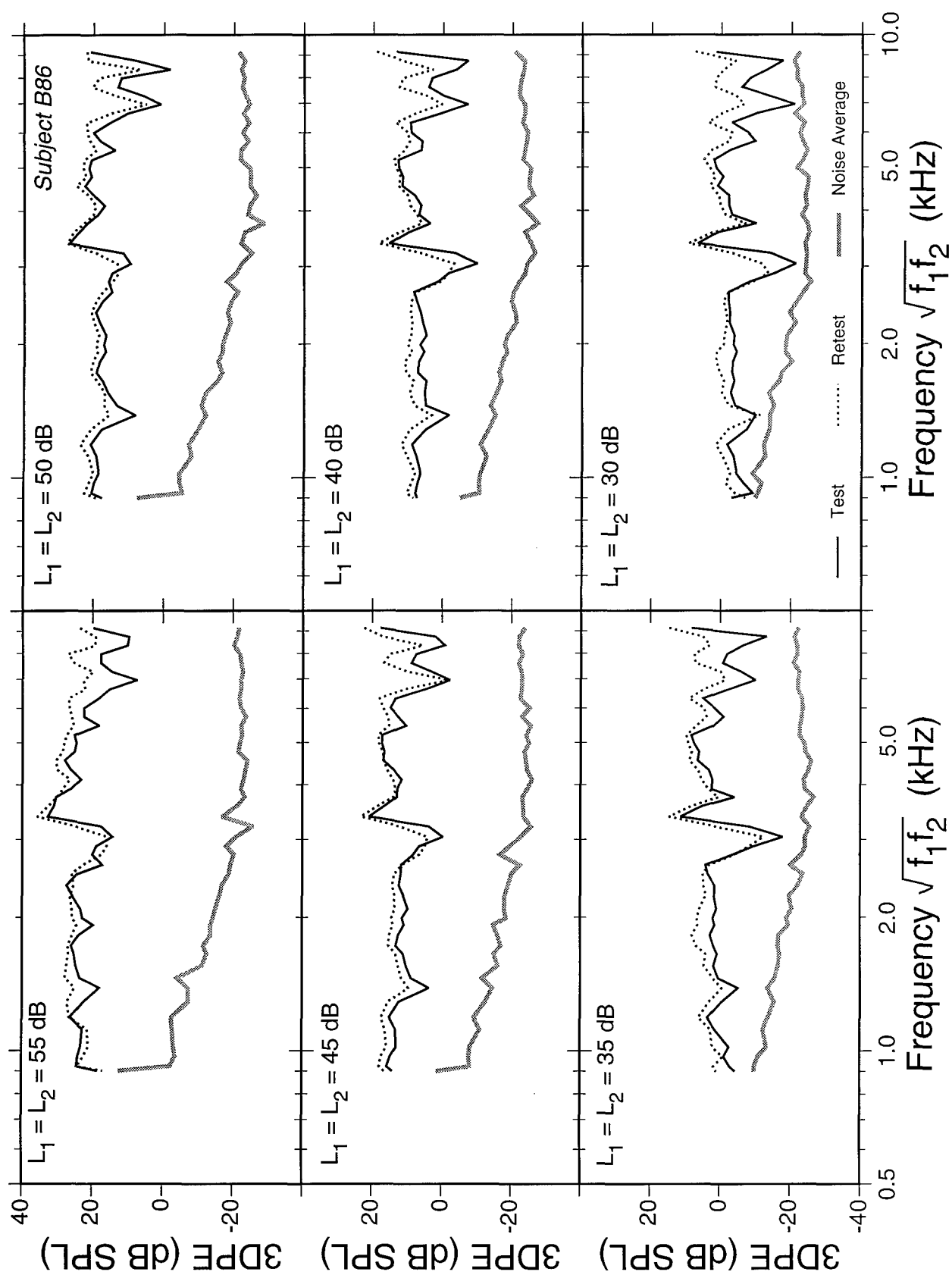


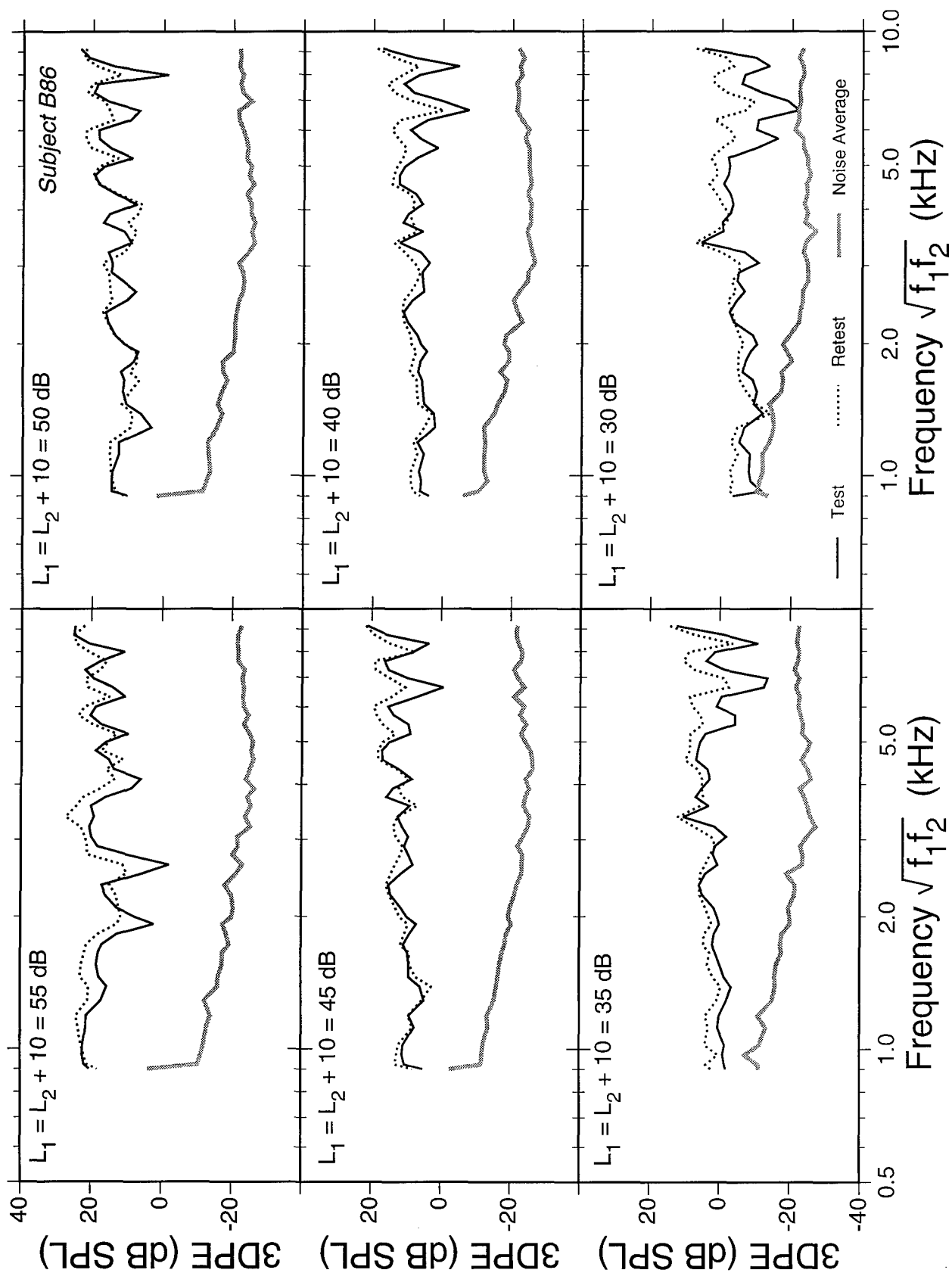


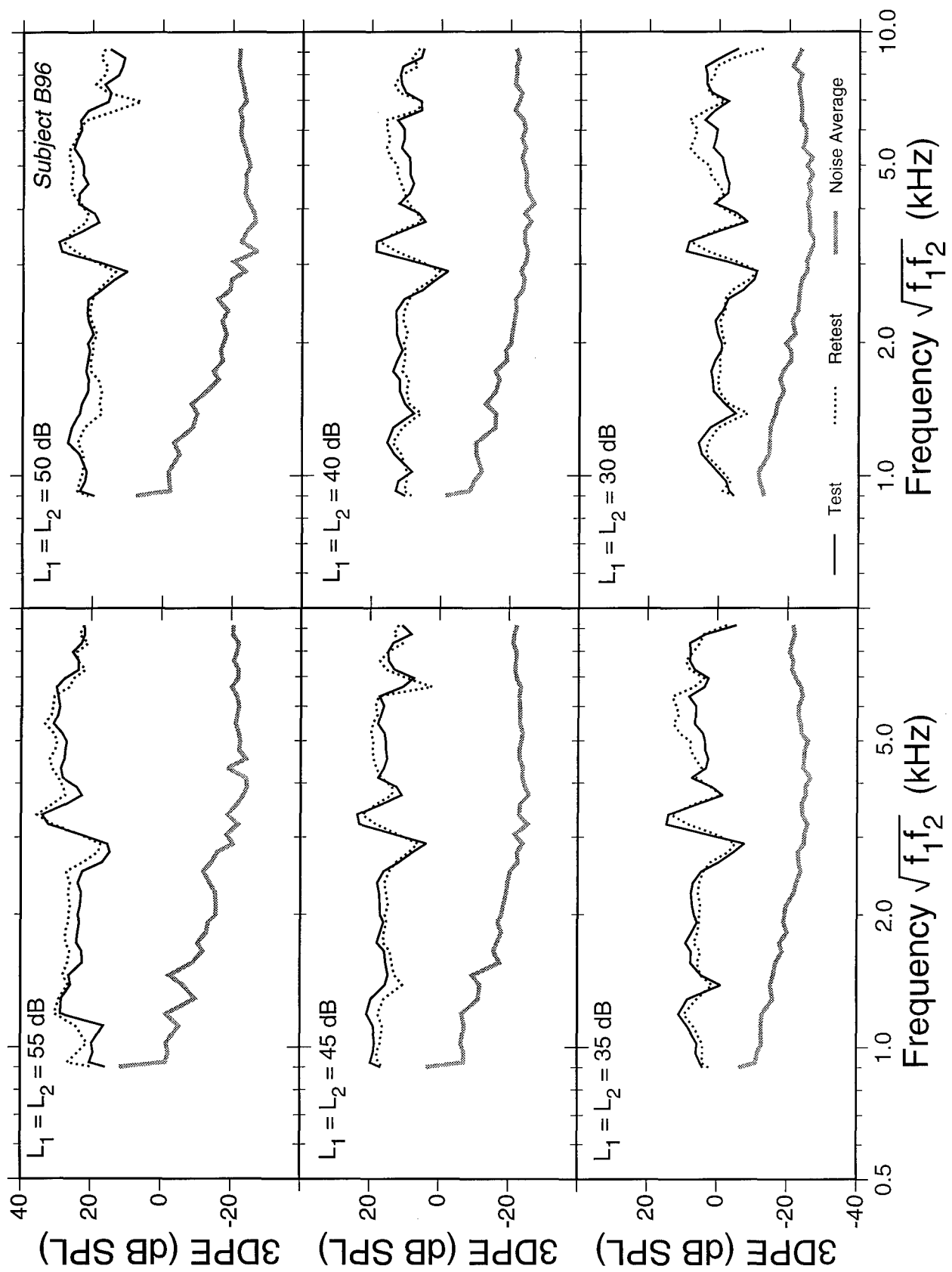


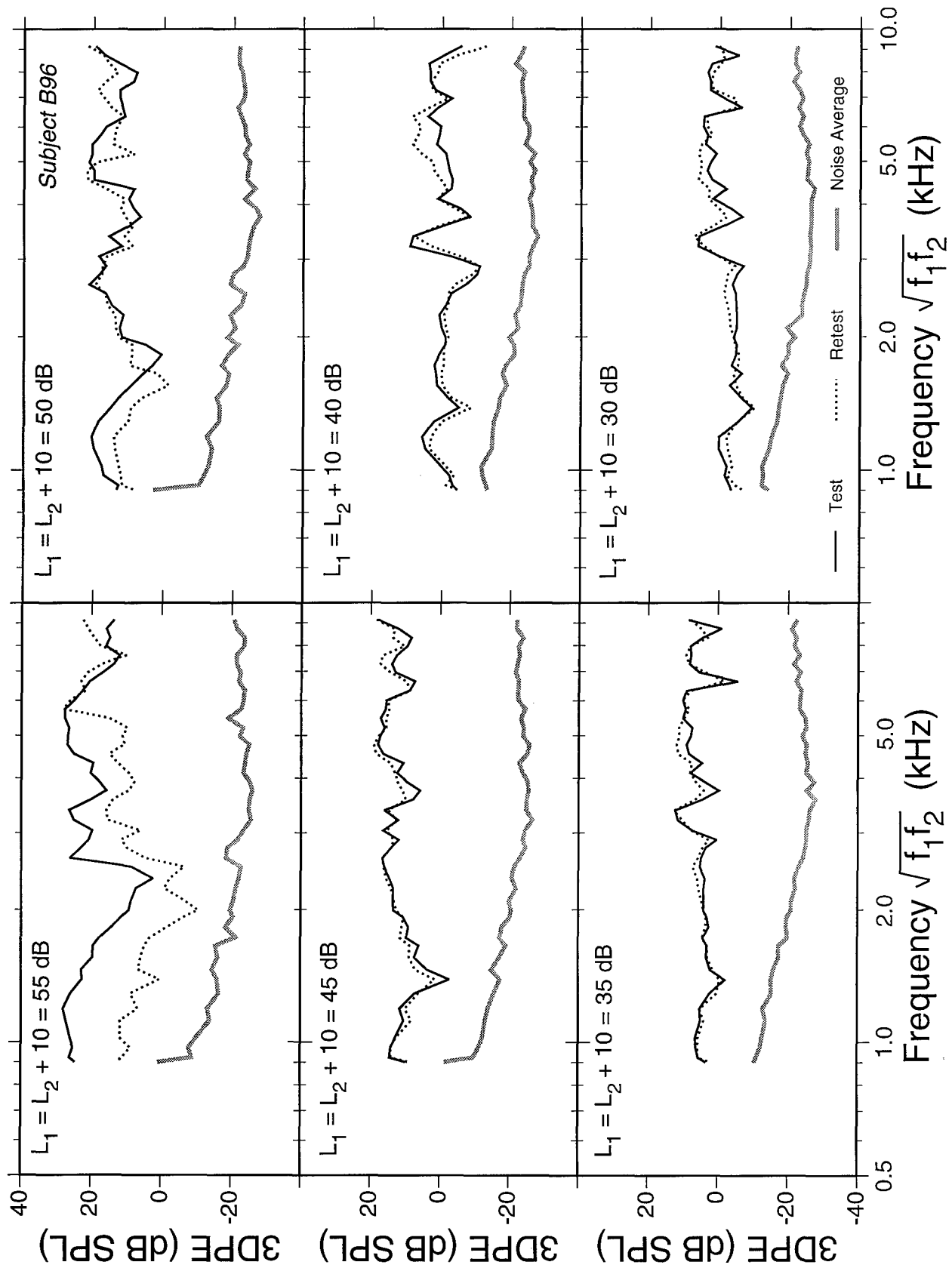




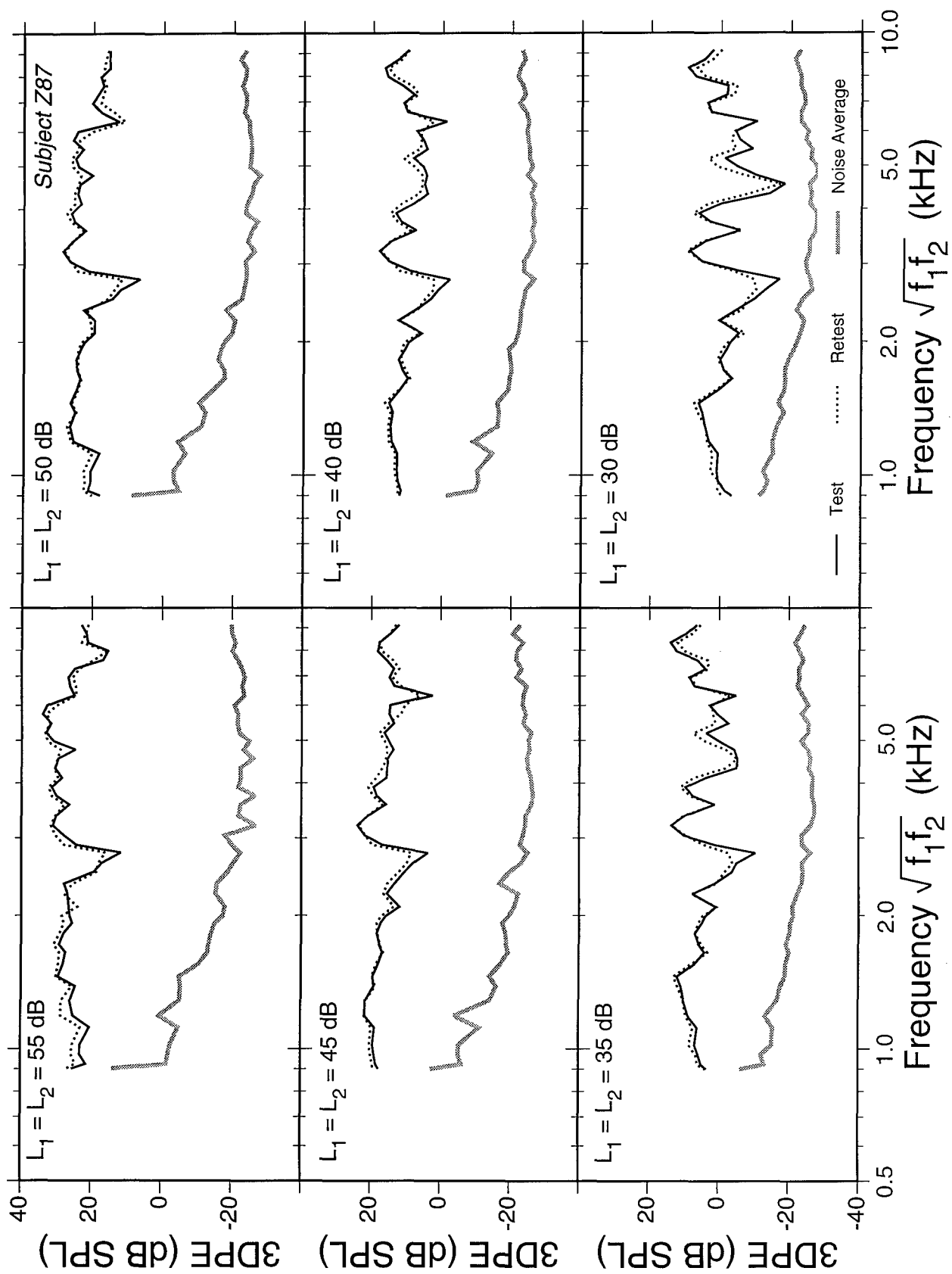


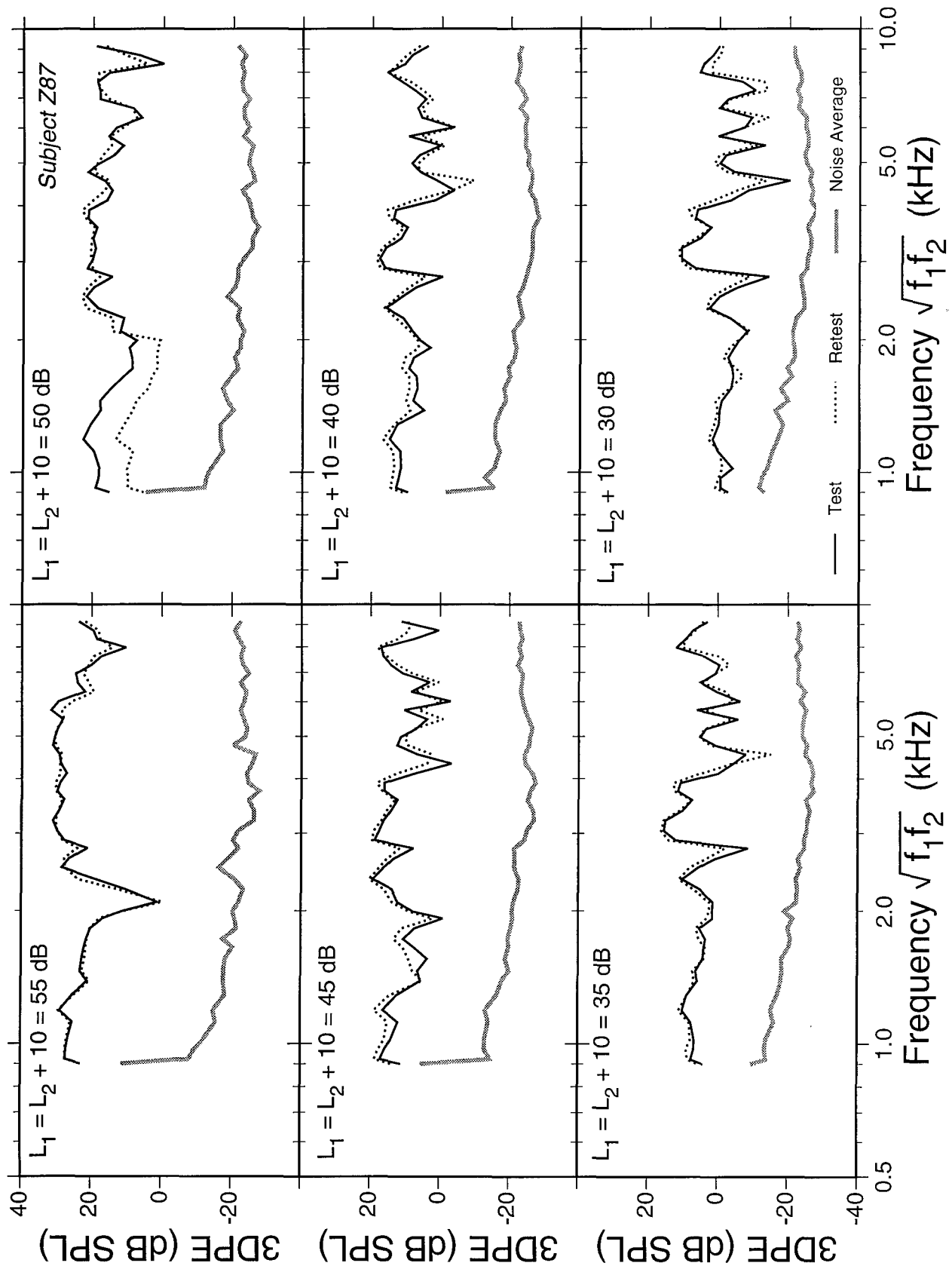












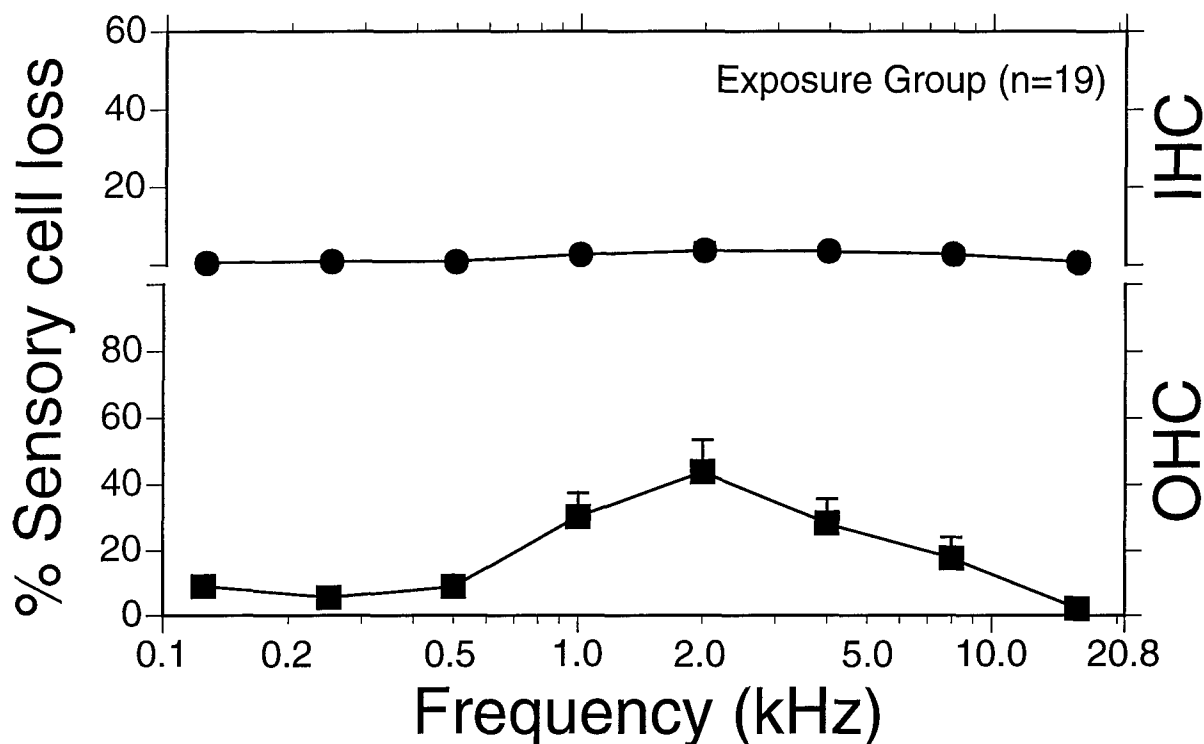
## APPENDIX C

Summary Data for the Group Exposed to:

### Conventional Shock Tube

~157 dB peak SPL, 12X, 3/minute

Animal #		
U18	-	Completed the Histology Protocol Only
X63	-	Completed the Histology Protocol Only
X64	-	Completed the Histology Protocol Only
X66	-	Completed the Histology Protocol Only
X68	-	Completed the Histology Protocol Only
X70	-	Completed the Histology Protocol Only
X76	-	Completed the Histology Protocol Only
X78	-	Completed the Histology Protocol Only
X85	-	Completed the Histology Protocol Only
X87	-	Completed the Histology Protocol Only
Z23	-	Completed the Histology Protocol Only
Z24	-	Completed the Histology Protocol Only
Z25	-	Completed the Histology Protocol Only
Z26	-	Completed the Histology Protocol Only
Z30	-	Completed the Histology Protocol Only
Z32	-	Completed the Histology Protocol Only
Z34	-	Completed the Histology Protocol Only
Z38	-	Completed the Histology Protocol Only
Z39	-	Completed the Histology Protocol Only



Group mean percent inner (●) and (■) outer hair cell loss from a group of animals exposed to 12 impulses of approximately 157 dB peak SPL at a rate of 3 impulses per minute. Error bars represent one standard error of the mean.

## Individual and Group Mean Histology Summary

Group summary data (means, standard deviations, and standard errors of the mean) are presented for total and percent cell losses measured in octave-band lengths of the cochlea on Pages 200 and 201. Following the summary data, individual animal total and percent cell losses are presented (Pages 202 through 213). Following the tabulated data, individual animal cochleograms are presented on Pages 214 through 232. The three graphs on these pages show: (top) a "standard" cochleogram showing percent inner and outer sensory cell losses; (middle) percent cell losses in each of the three rows of outer hair cells; and (bottom) percent missing supporting (pillar) cells.

Conventional Shock Tube  
~157 dB peak SPL, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Group means							
0.125 kHz	0.7	6.3	18.9	29.1	54.3	0.0	0.4
0.25 kHz	2.1	10.5	15.0	32.3	57.8	0.8	1.4
0.5 kHz	2.1	28.5	37.8	24.8	91.2	0.5	1.3
1 kHz	6.6	116.6	120.2	63.4	300.2	9.9	8.8
2 kHz	9.3	174.1	167.5	107.0	448.5	15.7	14.8
4 kHz	9.2	104.6	101.3	87.1	293.0	15.7	8.3
8 kHz	7.5	56.4	60.2	62.9	179.5	15.5	14.4
16 kHz	1.5	7.1	5.3	6.1	18.4	0.3	0.8
TOTALS	38.9	504.0	526.2	412.7	1442.9	58.4	50.2
Group standard deviations							
0.125 kHz	1.1	9.0	25.2	27.9	58.1	0.0	0.8
0.25 kHz	2.8	16.5	16.5	29.3	54.5	2.4	3.5
0.5 kHz	3.1	64.5	60.4	33.7	153.7	1.6	2.6
1 kHz	13.1	119.0	110.5	91.9	300.7	27.8	18.7
2 kHz	22.8	162.7	162.2	120.6	428.3	50.1	35.6
4 kHz	12.2	113.1	115.1	104.8	329.3	24.4	13.7
8 kHz	19.3	89.9	91.6	100.9	278.8	53.3	35.9
16 kHz	2.7	16.3	10.6	11.7	38.0	1.1	3.0
TOTALS	44.3	412.6	423.9	372.1	1175.9	92.1	73.4
Group standard errors							
0.125 kHz	0.3	2.1	5.8	6.4	13.3	0.0	0.2
0.25 kHz	0.7	3.8	3.8	6.7	12.5	0.6	0.8
0.5 kHz	0.7	14.8	13.9	7.7	35.3	0.4	0.6
1 kHz	3.0	27.3	25.3	21.1	69.0	6.4	4.3
2 kHz	5.2	37.3	37.2	27.7	98.3	11.5	8.2
4 kHz	2.8	25.9	26.4	24.0	75.5	5.6	3.1
8 kHz	4.4	20.6	21.0	23.1	64.0	12.2	8.2
16 kHz	0.6	3.7	2.4	2.7	8.7	0.3	0.7
TOTALS	10.2	94.7	97.3	85.4	269.8	21.1	16.8

Conventional Shock Tube  
~157 dB peak SPL, 12X, 3/minute

Percent sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Group means							
0.125 kHz	0.46	3.16	9.39	14.47	9.01	0.00	0.18
0.25 kHz	0.80	3.05	4.29	9.23	5.53	0.15	0.41
0.5 kHz	0.79	8.42	11.04	7.18	8.88	0.10	0.38
1 kHz	2.59	35.26	36.24	19.50	30.34	1.88	2.67
2 kHz	3.55	50.87	48.94	31.46	43.76	2.75	4.21
4 kHz	3.40	30.26	29.41	25.31	28.33	2.81	2.41
8 kHz	2.64	16.66	17.82	18.69	17.72	2.71	4.09
16 kHz	0.62	2.36	1.74	2.01	2.04	0.05	0.27
Group standard deviations							
0.125 kHz	0.76	4.60	12.75	13.81	9.75	0.00	0.40
0.25 kHz	1.07	4.88	4.88	8.54	5.37	0.46	1.01
0.5 kHz	1.22	19.31	18.03	9.98	15.31	0.30	0.76
1 kHz	5.21	36.67	34.11	28.96	31.29	5.31	5.80
2 kHz	8.58	47.36	47.18	35.61	41.78	8.73	10.04
4 kHz	4.51	32.32	33.09	30.33	31.56	4.32	3.87
8 kHz	6.78	26.71	27.24	30.12	27.66	9.32	10.15
16 kHz	1.08	5.59	3.55	3.94	4.29	0.23	0.94
Group standard errors							
0.125 kHz	0.17	1.06	2.92	3.17	2.24	0.00	0.09
0.25 kHz	0.24	1.12	1.12	1.96	1.23	0.10	0.23
0.5 kHz	0.28	4.43	4.14	2.29	3.51	0.07	0.18
1 kHz	1.19	8.41	7.82	6.64	7.18	1.22	1.33
2 kHz	1.97	10.87	10.82	8.17	9.58	2.00	2.30
4 kHz	1.04	7.41	7.59	6.96	7.24	0.99	0.89
8 kHz	1.56	6.13	6.25	6.91	6.35	2.14	2.33
16 kHz	0.25	1.28	0.81	0.90	0.98	0.05	0.22

Conventional Shock Tube  
~157 dB peak SPL, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla U18							
0.125 kHz	1	7	20	68	95	0	2
0.25 kHz	4	11	29	60	100	0	1
0.5 kHz	3	7	74	35	116	0	0
1 kHz	1	12	26	47	85	0	0
2 kHz	3	9	14	53	76	0	0
4 kHz	1	12	13	36	61	0	1
8 kHz	0	4	7	21	32	0	0
16 kHz	1	5	2	2	9	0	0
TOTALS	14	67	185	322	574	0	4
Chinchilla X63							
0.125 kHz	0	4	10	15	29	0	0
0.25 kHz	1	29	22	27	78	0	1
0.5 kHz	11	145	120	28	293	5	7
1 kHz	23	310	307	266	883	14	24
2 kHz	4	294	270	195	759	1	5
4 kHz	29	95	88	32	215	54	11
8 kHz	0	2	0	1	3	0	0
16 kHz	0	4	2	0	6	0	2
TOTALS	68	883	819	564	2266	74	50
Chinchilla X64							
0.125 kHz	4	34	97	82	213	0	0
0.25 kHz	10	73	76	98	247	10	15
0.5 kHz	8	259	257	149	665	5	7
1 kHz	48	321	321	318	960	111	78
2 kHz	3	327	323	275	925	0	1
4 kHz	1	141	185	163	489	0	2
8 kHz	0	25	42	38	105	0	2
16 kHz	0	7	3	3	13	0	0
TOTALS	74	1187	1304	1126	3617	126	105

Conventional Shock Tube  
~157 dB peak SPL, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla X66							
0.125 kHz	0	4	2	18	24	0	0
0.25 kHz	0	10	18	16	44	0	0
0.5 kHz	0	17	42	15	74	0	0
1 kHz	2	55	58	26	139	0	1
2 kHz	0	81	72	15	168	0	0
4 kHz	4	4	9	5	18	0	0
8 kHz	10	76	99	88	263	10	16
16 kHz	1	5	3	4	12	0	0
TOTALS	17	252	303	187	742	10	17
Chinchilla X68							
0.125 kHz	1	4	5	21	30	0	0
0.25 kHz	1	1	8	46	55	0	0
0.5 kHz	1	4	7	13	24	0	0
1 kHz	0	291	258	39	588	0	1
2 kHz	1	329	326	100	755	4	2
4 kHz	3	39	66	39	144	10	11
8 kHz	2	11	11	10	32	0	0
16 kHz	4	3	3	1	7	0	0
TOTALS	13	682	684	269	1635	14	14
Chinchilla X70							
0.125 kHz	1	23	45	75	143	0	0
0.25 kHz	1	3	13	112	128	0	0
0.5 kHz	2	21	26	66	113	0	3
1 kHz	0	216	143	67	426	0	4
2 kHz	7	339	324	245	908	2	42
4 kHz	3	113	111	94	318	5	7
8 kHz	2	20	31	22	73	0	0
16 kHz	0	0	1	0	1	0	0
TOTALS	16	735	694	681	2110	7	56



Conventional Shock Tube  
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Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla X76							
0.125 kHz	0	14	34	45	93	0	3
0.25 kHz	2	12	19	20	51	0	0
0.5 kHz	1	10	1	5	16	0	0
1 kHz	1	13	5	5	23	0	0
2 kHz	5	54	11	6	71	19	0
4 kHz	34	119	17	10	146	72	10
8 kHz	17	45	46	36	127	22	19
16 kHz	6	1	1	0	2	0	0
TOTALS	66	268	134	127	529	113	32
Chinchilla X78							
0.125 kHz	1	4	5	17	26	0	0
0.25 kHz	0	2	11	8	21	0	0
0.5 kHz	2	8	29	15	52	0	0
1 kHz	2	8	25	8	41	0	1
2 kHz	6	6	1	4	11	2	0
4 kHz	2	3	1	3	7	0	1
8 kHz	2	2	3	6	11	0	0
16 kHz	10	5	7	5	17	0	0
TOTALS	25	38	82	66	186	2	2
Chinchilla X85							
0.125 kHz	1	12	20	40	72	0	0
0.25 kHz	1	12	15	26	53	4	4
0.5 kHz	6	33	30	29	92	0	7
1 kHz	2	226	215	72	513	0	10
2 kHz	1	355	351	187	893	0	33
4 kHz	33	355	355	326	1036	66	56
8 kHz	84	293	301	296	890	234	155
16 kHz	1	0	1	2	3	0	0
TOTALS	129	1286	1288	978	3552	304	265

Conventional Shock Tube  
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Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla X87							
0.125 kHz	1	3	4	28	35	0	0
0.25 kHz	1	8	2	17	27	0	0
0.5 kHz	1	0	2	3	5	0	0
1 kHz	0	8	3	1	12	0	0
2 kHz	3	31	13	12	56	0	0
4 kHz	1	63	67	73	203	1	3
8 kHz	0	279	270	263	812	0	3
16 kHz	2	67	36	43	146	0	0
TOTALS	9	459	397	440	1296	1	6
Chinchilla Z23							
0.125 kHz	0	0	1	1	2	0	0
0.25 kHz	0	7	7	10	24	0	1
0.5 kHz	1	10	20	26	56	0	0
1 kHz	0	7	19	30	56	0	0
2 kHz	0	4	2	5	11	0	0
4 kHz	1	4	3	6	13	0	0
8 kHz	0	0	2	3	5	0	0
16 kHz	0	1	1	0	2	0	1
TOTALS	2	33	55	81	169	0	2
Chinchilla Z24							
0.125 kHz	0	0	4	6	10	0	1
0.25 kHz	0	5	4	8	17	0	0
0.5 kHz	3	10	37	10	57	0	0
1 kHz	7	8	49	11	68	2	0
2 kHz	7	47	37	27	111	0	2
4 kHz	9	31	20	18	69	19	2
8 kHz	1	5	3	9	17	0	0
16 kHz	0	0	2	4	6	0	0
TOTALS	27	106	156	93	355	21	5

Conventional Shock Tube  
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Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla Z25							
0.125 kHz	0	2	1	7	10	0	0
0.25 kHz	3	6	15	44	65	0	0
0.5 kHz	0	2	15	27	44	0	0
1 kHz	7	177	210	191	578	4	7
2 kHz	13	332	332	331	995	9	7
4 kHz	9	319	331	332	982	8	15
8 kHz	0	150	185	294	629	0	4
16 kHz	0	1	0	11	12	0	0
TOTALS	32	989	1089	1237	3315	21	33
Chinchilla Z26							
0.125 kHz	0	0	4	2	6	0	0
0.25 kHz	2	4	14	24	42	0	2
0.5 kHz	0	4	9	15	28	0	1
1 kHz	1	180	184	29	393	0	3
2 kHz	0	17	27	23	67	0	0
4 kHz	0	1	5	12	18	0	0
8 kHz	2	96	63	47	206	3	8
16 kHz	4	33	34	33	100	5	13
TOTALS	9	335	340	185	860	8	27
Chinchilla Z30							
0.125 kHz	0	0	26	8	34	0	0
0.25 kHz	9	2	14	15	31	1	2
0.5 kHz	0	1	11	8	20	0	0
1 kHz	0	231	241	7	479	0	3
2 kHz	2	361	361	52	774	0	6
4 kHz	0	166	169	124	459	0	0
8 kHz	16	21	23	21	65	20	16
16 kHz	0	0	0	0	0	0	0
TOTALS	27	782	845	235	1862	21	27

Conventional Shock Tube  
~157 dB peak SPL, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla Z32							
0.125 kHz	3	1	8	10	19	0	0
0.25 kHz	3	0	1	4	5	0	0
0.5 kHz	0	0	2	1	3	0	0
1 kHz	0	0	0	1	1	0	0
2 kHz	0	1	2	1	4	0	1
4 kHz	8	27	25	26	78	12	10
8 kHz	1	3	1	2	6	0	0
16 kHz	0	0	0	0	0	0	0
TOTALS	15	32	39	45	116	12	11
Chinchilla Z34							
0.125 kHz	0	0	3	8	11	0	1
0.25 kHz	1	3	2	24	29	0	1
0.5 kHz	0	2	15	14	31	0	0
1 kHz	31	80	112	59	251	57	30
2 kHz	101	355	355	350	1060	218	152
4 kHz	30	222	208	150	580	4	28
8 kHz	4	31	52	31	114	5	5
16 kHz	0	2	4	6	12	0	0
TOTALS	167	695	751	642	2088	284	217
Chinchilla Z38							
0.125 kHz	0	7	9	20	36	0	0
0.25 kHz	1	9	4	25	38	0	0
0.5 kHz	0	6	7	4	17	0	0
1 kHz	0	6	23	7	36	0	0
2 kHz	1	5	14	6	25	0	0
4 kHz	0	9	4	2	15	0	0
8 kHz	0	5	0	6	11	0	0
16 kHz	0	0	0	1	1	0	0
TOTALS	2	47	61	71	179	0	0

Conventional Shock Tube  
~157 dB peak SPL, 12X, 3/minute

Total sensory cell losses over octave band frequencies

	Inner	1st row	2nd row	3rd row	Comb.	Inner	Outer
	hair	outer	outer	outer	outer	pillar	pillar
	cells	hair	hair	hair	hair	cells	cells
	cells	cells	cells	cells	cells	cells	cells
Chinchilla Z39							
0.125 kHz	0	0	62	82	144	0	0
0.25 kHz	0	3	11	29	43	0	0
0.5 kHz	0	3	15	9	27	0	0
1 kHz	0	66	85	21	172	0	5
2 kHz	20	360	347	146	853	44	30
4 kHz	6	265	247	204	716	47	0
8 kHz	1	3	4	2	9	0	46
16 kHz	0	0	0	0	0	0	0
TOTALS	27	700	771	493	1964	91	81

Conventional Shock Tube  
~157 dB peak SPL, 12X, 3/minute

Percent sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla U18							
0.125 kHz	0.7	3.5	10.0	33.8	15.8	0.0	1.0
0.25 kHz	1.5	3.1	8.2	17.0	9.4	0.0	0.3
0.5 kHz	1.1	2.0	21.0	9.9	11.0	0.0	0.0
1 kHz	0.4	3.6	7.8	14.0	8.5	0.0	0.0
2 kHz	1.2	2.6	4.1	15.5	7.4	0.0	0.0
4 kHz	0.4	3.5	3.8	10.5	5.9	0.0	0.3
8 kHz	0.0	1.2	2.0	6.1	3.1	0.0	0.0
16 kHz	0.4	1.6	0.7	0.7	1.0	0.0	0.0
Chinchilla X63							
0.125 kHz	0.0	2.1	5.3	8.0	5.1	0.0	0.0
0.25 kHz	0.4	8.9	6.7	8.3	8.0	0.0	0.3
0.5 kHz	4.5	44.3	36.7	8.6	29.9	1.0	2.1
1 kHz	9.5	99.7	98.7	85.5	94.6	2.8	7.7
2 kHz	1.7	92.5	84.9	61.3	79.6	0.2	1.6
4 kHz	11.7	29.9	27.7	10.1	22.6	10.5	3.5
8 kHz	0.0	0.6	0.0	0.3	0.3	0.0	0.0
16 kHz	0.0	1.4	0.7	0.0	0.7	0.0	0.7
Chinchilla X64							
0.125 kHz	2.7	17.7	50.5	42.7	37.0	0.0	0.0
0.25 kHz	3.9	21.5	22.4	28.9	24.3	1.9	4.4
0.5 kHz	3.1	76.9	76.3	44.2	65.8	0.9	2.1
1 kHz	19.4	100.0	100.0	99.1	99.7	21.5	24.3
2 kHz	1.2	100.0	98.8	84.1	94.3	0.0	0.3
4 kHz	0.4	43.0	56.4	49.7	49.7	0.0	0.6
8 kHz	0.0	7.6	12.8	11.6	10.7	0.0	0.6
16 kHz	0.0	2.4	1.0	1.0	1.5	0.0	0.0
Chinchilla X66							
0.125 kHz	0.0	2.1	1.1	9.5	4.2	0.0	0.0
0.25 kHz	0.0	3.0	5.4	4.8	4.4	0.0	0.0
0.5 kHz	0.0	5.1	12.7	4.5	7.4	0.0	0.0
1 kHz	0.8	17.5	18.4	8.3	14.7	0.0	0.3
2 kHz	0.0	25.1	22.3	4.6	17.3	0.0	0.0
4 kHz	1.6	1.2	2.8	1.6	1.9	0.0	0.0
8 kHz	3.9	23.6	30.7	27.3	27.2	1.9	5.0
16 kHz	0.4	1.7	1.0	1.4	1.4	0.0	0.0

Conventional Shock Tube  
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Percent sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla X68							
0.125 kHz	0.7	2.1	2.6	10.9	5.2	0.0	0.0
0.25 kHz	0.4	0.3	2.4	13.6	5.4	0.0	0.0
0.5 kHz	0.4	1.2	2.1	3.8	2.4	0.0	0.0
1 kHz	0.0	90.7	80.4	12.1	61.1	0.0	0.3
2 kHz	0.4	100.0	99.1	30.4	76.5	0.8	0.6
4 kHz	1.2	11.9	20.1	11.9	14.6	1.9	3.4
8 kHz	0.8	3.4	3.4	3.1	3.3	0.0	0.0
16 kHz	1.7	1.0	1.0	0.3	0.8	0.0	0.0
Chinchilla X70							
0.125 kHz	0.7	11.6	22.6	37.7	24.0	0.0	0.0
0.25 kHz	0.4	0.9	3.7	32.1	12.2	0.0	0.0
0.5 kHz	0.8	6.0	7.4	18.9	10.8	0.0	0.9
1 kHz	0.0	65.3	43.2	20.2	42.9	0.0	1.2
2 kHz	2.8	100.0	95.6	72.3	89.3	0.4	12.4
4 kHz	1.1	33.3	32.7	27.7	31.2	0.9	2.1
8 kHz	0.7	5.9	9.2	6.5	7.2	0.0	0.0
16 kHz	0.0	0.0	0.3	0.0	0.1	0.0	0.0
Chinchilla X76							
0.125 kHz	0.0	6.7	16.2	21.4	14.8	0.0	1.4
0.25 kHz	0.7	3.2	5.1	5.4	4.6	0.0	0.0
0.5 kHz	0.4	2.7	0.3	1.4	1.5	0.0	0.0
1 kHz	0.4	3.7	1.4	1.4	2.2	0.0	0.0
2 kHz	1.9	15.1	3.1	1.7	6.6	3.3	0.0
4 kHz	12.2	33.2	4.7	2.8	13.6	12.5	2.8
8 kHz	5.9	12.6	12.8	10.1	11.8	3.8	5.3
16 kHz	2.3	0.3	0.3	0.0	0.2	0.0	0.0
Chinchilla X78							
0.125 kHz	0.6	2.0	2.4	8.3	4.2	0.0	0.0
0.25 kHz	0.0	0.6	3.1	2.2	2.0	0.0	0.0
0.5 kHz	0.7	2.2	8.1	4.2	4.8	0.0	0.0
1 kHz	0.8	2.3	7.3	2.3	4.0	0.0	0.3
2 kHz	2.3	1.7	0.3	1.1	1.0	0.4	0.0
4 kHz	0.7	0.9	0.3	0.9	0.7	0.0	0.3
8 kHz	0.7	0.6	0.9	1.7	1.1	0.0	0.0
16 kHz	4.0	1.6	2.2	1.6	1.8	0.0	0.0

Conventional Shock Tube  
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Percent sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla X85							
0.125 kHz	0.6	5.8	9.6	19.2	11.5	0.0	0.0
0.25 kHz	0.4	3.3	4.1	7.1	4.8	0.7	1.1
0.5 kHz	2.2	9.0	8.2	7.9	8.4	0.0	1.9
1 kHz	0.7	65.1	62.0	20.7	49.3	0.0	2.9
2 kHz	0.4	100.0	98.9	52.7	83.9	0.0	9.3
4 kHz	11.9	100.0	100.0	91.8	97.3	11.5	15.8
8 kHz	29.6	83.0	85.3	83.9	84.1	40.9	43.9
16 kHz	0.4	0.0	0.3	0.6	0.3	0.0	0.0
Chinchilla X87							
0.125 kHz	0.7	1.6	2.1	14.7	6.1	0.0	0.0
0.25 kHz	0.4	2.4	0.6	5.1	2.7	0.0	0.0
0.5 kHz	0.4	0.0	0.6	0.9	0.5	0.0	0.0
1 kHz	0.0	2.5	0.9	0.3	1.2	0.0	0.0
2 kHz	1.2	9.6	4.0	3.7	5.8	0.0	0.0
4 kHz	0.4	19.6	20.8	22.7	21.0	0.2	0.9
8 kHz	0.0	86.6	83.9	81.7	84.1	0.0	0.9
16 kHz	0.9	23.3	12.5	14.9	16.9	0.0	0.0
Chinchilla Z23							
0.125 kHz	0.0	0.0	0.5	0.5	0.3	0.0	0.0
0.25 kHz	0.0	2.0	2.0	2.9	2.3	0.0	0.3
0.5 kHz	0.4	2.9	5.8	7.6	5.4	0.0	0.0
1 kHz	0.0	2.2	5.8	9.2	5.7	0.0	0.0
2 kHz	0.0	1.2	0.6	1.5	1.1	0.0	0.0
4 kHz	0.4	1.2	0.9	1.8	1.3	0.0	0.0
8 kHz	0.0	0.0	0.6	0.9	0.5	0.0	0.0
16 kHz	0.0	0.3	0.3	0.0	0.2	0.0	0.3
Chinchilla Z24							
0.125 kHz	0.0	0.0	1.9	2.8	1.6	0.0	0.5
0.25 kHz	0.0	1.4	1.1	2.2	1.6	0.0	0.0
0.5 kHz	1.1	2.7	10.0	2.7	5.1	0.0	0.0
1 kHz	2.6	2.3	14.0	3.1	6.5	0.4	0.0
2 kHz	2.6	13.1	10.3	7.5	10.3	0.0	0.6
4 kHz	3.2	8.7	5.6	5.0	6.4	3.3	0.6
8 kHz	0.3	1.4	0.8	2.5	1.6	0.0	0.0
16 kHz	0.0	0.0	0.6	1.3	0.6	0.0	0.0



Conventional Shock Tube  
~157 dB peak SPL, 12X, 3/minute

Percent sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla Z25							
0.125 kHz	0.0	1.0	0.5	3.6	1.7	0.0	0.0
0.25 kHz	1.2	1.7	4.4	12.8	6.3	0.0	0.0
0.5 kHz	0.0	0.6	4.4	7.9	4.3	0.0	0.0
1 kHz	2.8	54.3	64.4	58.6	59.1	0.8	2.1
2 kHz	5.2	100.0	100.0	99.7	99.9	1.7	2.1
4 kHz	3.4	96.1	99.7	100.0	98.6	1.5	4.5
8 kHz	0.0	45.3	55.9	88.8	63.3	0.0	1.2
16 kHz	0.0	0.3	0.0	3.7	1.3	0.0	0.0
Chinchilla Z26							
0.125 kHz	0.0	0.0	1.9	1.0	1.0	0.0	0.0
0.25 kHz	0.7	1.1	3.8	6.6	3.8	0.0	0.5
0.5 kHz	0.0	1.1	2.5	4.1	2.6	0.0	0.3
1 kHz	0.4	52.0	53.2	8.4	37.9	0.0	0.9
2 kHz	0.0	4.8	7.6	6.5	6.3	0.0	0.0
4 kHz	0.0	0.3	1.4	3.4	1.7	0.0	0.0
8 kHz	0.7	27.2	17.8	13.3	19.4	0.5	2.3
16 kHz	1.6	10.4	10.8	10.4	10.5	1.0	4.1
Chinchilla Z30							
0.125 kHz	0.0	0.0	12.3	3.8	5.4	0.0	0.0
0.25 kHz	3.2	0.5	3.8	4.0	2.8	0.2	0.5
0.5 kHz	0.0	0.3	3.0	2.2	1.8	0.0	0.0
1 kHz	0.0	65.4	68.3	2.0	45.2	0.0	0.8
2 kHz	0.7	100.0	100.0	14.4	71.5	0.0	1.7
4 kHz	0.0	46.0	46.8	34.3	42.4	0.0	0.0
8 kHz	5.5	5.8	6.4	5.8	6.0	3.4	4.4
16 kHz	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chinchilla Z32							
0.125 kHz	2.1	0.5	4.2	5.2	3.3	0.0	0.0
0.25 kHz	1.2	0.0	0.3	1.2	0.5	0.0	0.0
0.5 kHz	0.0	0.0	0.6	0.3	0.3	0.0	0.0
1 kHz	0.0	0.0	0.0	0.3	0.1	0.0	0.0
2 kHz	0.0	0.3	0.6	0.3	0.4	0.0	0.3
4 kHz	3.1	8.3	7.7	8.0	8.0	2.3	3.1
8 kHz	0.4	0.9	0.3	0.6	0.6	0.0	0.0
16 kHz	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Conventional Shock Tube  
~157 dB peak SPL, 12X, 3/minute

Percent sensory cell losses over octave band frequencies

	Inner hair cells	1st row outer hair cells	2nd row outer hair cells	3rd row outer hair cells	Comb. outer hair cells	Inner pillar cells	Outer pillar cells
Chinchilla Z34							
0.125 kHz	0.0	0.0	1.4	3.8	1.7	0.0	0.5
0.25 kHz	0.4	0.8	0.5	6.5	2.6	0.0	0.3
0.5 kHz	0.0	0.5	4.1	3.8	2.8	0.0	0.0
1 kHz	11.5	23.0	32.2	17.0	24.1	10.2	8.6
2 kHz	38.1	100.0	100.0	98.6	99.5	38.0	42.8
4 kHz	10.8	62.4	58.4	42.1	54.3	0.7	7.9
8 kHz	1.4	8.7	14.6	8.7	10.7	0.9	1.4
16 kHz	0.0	0.6	1.3	1.9	1.3	0.0	0.0
Chinchilla Z38							
0.125 kHz	0.0	3.4	4.4	9.8	5.9	0.0	0.0
0.25 kHz	0.4	2.5	1.1	7.0	3.5	0.0	0.0
0.5 kHz	0.0	1.7	2.0	1.1	1.6	0.0	0.0
1 kHz	0.0	1.8	6.7	2.1	3.5	0.0	0.0
2 kHz	0.4	1.4	4.0	1.7	2.4	0.0	0.0
4 kHz	0.0	2.6	1.1	0.6	1.4	0.0	0.0
8 kHz	0.0	1.4	0.0	1.7	1.0	0.0	0.0
16 kHz	0.0	0.0	0.0	0.3	0.1	0.0	0.0
Chinchilla Z39							
0.125 kHz	0.0	0.0	29.0	38.3	22.4	0.0	0.0
0.25 kHz	0.0	0.8	2.9	7.7	3.8	0.0	0.0
0.5 kHz	0.0	0.8	4.0	2.4	2.4	0.0	0.0
1 kHz	0.0	18.6	23.9	5.9	16.1	0.0	1.4
2 kHz	7.4	99.2	95.6	40.2	78.3	7.5	8.3
4 kHz	2.1	72.8	67.9	56.0	65.6	8.0	0.0
8 kHz	0.3	0.8	1.1	0.6	0.8	0.0	12.7
16 kHz	0.0	0.0	0.0	0.0	0.0	0.0	0.0

